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The product of a professional workshop, 10 papers discuss brain damage. An introduction to clinical neuropsychology is presented by H. Carl Haywood. A section on neurological foundations includes papers on the organization of the central nervous system by Jack T. Tapp and Lance L. Simpson, on epilepsy by Angela T. Folsom, and on organic language disorders in children by Kathryn Barth Horton. Aspects of psychological diagnosis considered are the use of psychological tests in diagnosis by Homer B. C. Reed, Jr., and screening children through the laboratory method by Luciano L'Abate. Approaches to treatment are included in a discussion of educators and minimal brain dysfunction by Lloyd M. Dunn, in a summary of the literature on behavior disorders in brain injured children by Gary M. Clark, and in a review of the theories and methods of Doman and Delacato by Charles W. McDonald. An annotated bibliography on anoxia by Harry Lewis is also included. (RJ)

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brain damage in school age children

editor

h. carl haywood

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THE COUNCIL FOR EXCEPTIONAL CHILDREN

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Brain Damage in School Age Children

H. Carl Haywood, Editor

U.S. DEPARTMENT OF HEALTH, EDUCATION & WELFARE
OFFICE OF EDUCATION

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Foreword

Since its inception in 1949, the Southern Regional Education Board (SREB) has in various ways been committed to the needs of exceptional children. The Board has shown its awareness via its assignment of part and full time professional staff and financial outlays in several projects.

SREB was established by interstate compact as a public agency and now includes fifteen member states cooperating to improve higher education. Member states are Alabama, Arkansas, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, North Carolina, Oklahoma, Tennessee, Texas, South Carolina, Virginia, and West Virginia. SREB conducts cooperative programs across state lines aimed at providing better graduate, professional, and technical education in the South. Some of its activities include studying the South's problems and needs in higher education; finding ways of solving these problems through region wide cooperation; administering student exchange programs between states and institutions; serving as an information center on educational activities in the region; and providing consultant services to states and institutions on problems related to higher education. The SREB has no coercive power and its success depends entirely on the interest and cooperation of participating states and institutions. Board membership consists of the governor of each compact state and four other persons, one of whom must be a legislator and one an educator appointed by the governor. SREB is supported by state, federal, foundation, and other funds.

In 1963, SREB received from the Office of Education funds for a three year project (Number 32-20-7180-1017) entitled, A Regional Approach to the Education and Training of Handicapped Children. This seminar illustrates one type of regional activity. The aim of the project was to seek advice and plan and implement activities which could best be done on a regional basis.

The content of this publication represents the subject matter presented at a regional seminar in the summer of 1965. At this time there was a large group of professional individuals in state departments and on college faculties who were concerned with the psychological diagnosis of brain damage in school age children. There was a limited amount of information available. The seminar tried to focus in on this problem; the results are partially available in this publication and in the diagnostic procedures being done in the SREB region. Each state's participants were asked to disseminate the seminar's content to the concerned professionals in their respective states.

Stanley E. Fudell
Lubbock, Texas

Preface

Any work which attempts to encompass the subject of brain damage in school age children would certainly be ambitious, and would probably be premature. The papers in this collection represent an effort to begin what the authors hope will be a long and fruitful discussion among those in the several disciplines which are directly concerned with the diagnosis and educational treatment of brain damaged children. As should be apparent, the papers have been written primarily with the special educator in mind, but it is hoped that they will prove to be useful also to teachers, psychologists, pediatricians, specialists in hearing and speech, child psychiatrists, and others whose work and concerns cause them to be confronted with the problems discussed here. It would clearly be impossible to write satisfactorily for such diverse consumption. With these limitations in mind, the authors have tried to consider the overall problem within the context of the main task of childhood, i.e., the educational enterprise.

These papers are the product of a professional workshop on brain damage in school age children, which was held at George Peabody College for Teachers in August of 1965, under the sponsorship and with the support of the Southern Regional Education Board. The richness of verbal interchange which characterizes such a meeting cannot be conveyed adequately by a mere presentation of the formal papers. To do so would require an exhaustive transcript of the informal conversations, the questions and answers, and the arguments that constitute one of the chief benefits of a professional workshop. At the same time, inclusion of these conversations would defeat one of the purposes of this volume, i.e., to present compactly and economically a summary of the thinking of the workshop staff. From the beginning, it was obvious that some limitation of the scope of the conference would be necessary. The director of this project and the Southern Regional Education Board chose to emphasize diagnostic problems rather than treatment problems. It seemed at the time, and still seems, that the chaotic state of research and programing in the area of educational treatment reflects in part the almost equally chaotic state of diagnostic efforts. The resolution of the former will depend upon substantial progress in the latter.

Readers will unquestionably wonder about the organization of the volume. While no papers of questionable relevance have been included, it is certain that these papers do not exhaust the supply of worthwhile thinking and research in this area. The volume is rather arbitrarily divided into five sections. The first section consists of a single paper, "An Introduction to Clinical Neuropsychology." An emphasis that is largely neurological is represented in the second section, which includes the papers by Tapp and Simpson, Horton, and Folsom. In the third section, consisting of the papers by Reed and L'Abate, the emphasis is on psychological diagnosis. A consideration of different approaches to treatment is represented in the fourth section, which includes the papers by Dunn, Clark, and McDonald. Finally, the appendix by Lewis is a consideration of the effects of cerebral anoxia. This last topic, while lacking the generality of some of the other papers, is included because of the intense interest that workers across several disciplines have had in this particular etiologic condition. Naturally, the editor recommends that the volume be read in the order in which the papers are presented. In this way, the reader who may not have an extensive background in the neurological sciences or in psychology can gain a cumulative sophistication as he progresses—sophistication that will be necessary in order to extract the fullest implications of the later chapters.

Many persons make a conference, and sometimes even more persons make a book. That is certainly the case with this work. The editor is grateful to the authors of these papers for their scholarship and indulgence. Particular gratitude is due the Southern Regional Education Board for its financial support and for the encouragement of Dr. Stanley Fudell, Director of the Handicapped Children's Project. Finally, the editor is grateful to Joanne Pegg and Patricia Mundy for their assistance in organizing and carrying out the workshop, and to Nancy Haywood for assisting in editing the manuscripts.

H. Carl Haywood

Part 1:

Introduction

Introduction to Clinical Neuropsychology

H. CARL HAYWOOD

Neuropsychology is the science which studies brain behavior relationships. Clinical neuropsychology is that branch of the science which relates behavior to pathological conditions of the nervous system, as such conditions occur in human beings. Both the basic science and its clinical application require the primary assumption that the brain is the principal mediating organ of behavior, an assumption whose tenability seems apparent today.

The nature of clinical neuropsychological inference is in some respects identical to the nature of clinical inference in the more general situation. In each case, the clinician takes a sample of the behavioral repertoire of an individual, and frequently assumes that his sample of that individual's behavior is representative of the subject's behavior. (The principal difference is that the neuropsychologist usually wishes to elicit the patient's best possible performance, rather than his typical performance.) Having drawn his sample, usually with the aid of standard diagnostic instruments, the clinician makes assumptions not only about the subject's behavior in situations which he has not observed, but he also makes inferences regarding processes which are unobservable. Thus, he assumes varying degrees of relationship between observable behavior and unobservable processes.

In the area of clinical neuropsychology, one of the jobs of the clinician is to make inferences regarding the condition of the brain from behavior which the brain presumably mediates. If the brain is indeed the principal mediating organ of behavior, and if there is a high degree of consistency across individuals in the functional anatomy of the brain, then it should be possible to establish lawful relationships involving the relative integrity of the central nervous system on the one hand and standard forms of behavior on the other. In modern times, pioneering efforts toward establishing such lawful brain behavior relationships have been made by such persons as Kurt Goldstein (e.g., 1923, 1924), D. O. Hebb (e.g., 1939, 1942, 1945; Hebb and Penfield, 1940), Wilder Penfield (e.g., Penfield and Roberts, 1959), Ward Halstead (1947), and Ralph Reitan (1962, 1966), among others.

Much of the modern work in clinical neuropsychology depends upon the assumption that there is considerable localization of function within the central nervous system, i.e., specific structures in reasonably constant locations perform predictable functions with respect to specifiable behaviors. As early as 1861, Broca had discovered some localization of function in the central nervous system, i. e., a speech center was located in the left cerebral hemisphere in the area of the third temporal convolution. Even more important work in functional neurology and neuroanatomy was contributed by Sherrington (e.g., 1933). In spite of these advances in relating specific behavior to the integrity of specific structures in the nervous system, Karl Lashley was able to formulate his famous "principle of mass action" (1926, 1929, 1935), working with infrahuman organisms. The principle of mass action stated that the behavioral deficit attributable to extirpation of central nervous system tissue is directly proportional to the amount of tissue removed. This principle appeared to be embarrassing to the specific localization position; however, Lashley was also aware of the principles of vicarious functioning and equipotentiality. In a limited sense, both these principles implied some localization of function even though localization was not held to be exclusive. Even the early work of Hebb in neuropsychology appeared to indicate that large portions of the central nervous system are unnecessary for normal functioning in the human adult. It is in the work of Wilder Penfield and his associates, and Ward Halstead and his students, that behavioral localization has been significantly advanced. Because of such work it is now possible to make reliable generalizations regarding the effects of focal brain lesions upon specific forms of observable and measurable behavior.

In addition to the necessary assumption of some localization of function, it will be useful to bear in mind that the brain is a complex system of interconnecting relays whose function is communication. Because of this peculiar nature of the nervous system, it is reasonable to assume that injury to any part of the communication network will impair the overall efficiency of the system, at least for a time.

The Concept of Brain Damage

Since brain damage is the subject matter of clinical neuropsychology, it will be useful to arrive at some definition of that term. The use of the term brain damage is not generally restricted to traumatic assault upon the brain, but may include also underdevelopment, malformation, and disease. At least nine kinds of brain disorders are usually included under the general heading of brain damage. These include the following: physical trauma, metabolic dysfunction, toxicity, degenerative brain disease, demyelinating disease, malformations, cerebral vascular disease, convulsive disorders, and neoplasms. In addition to considering the location of a particular brain lesion, it is beginning to be apparent that each of these particular etiological conditions may produce its peculiar and identifiable behavioral consequences.

It is generally agreed that brain damage in an individual produces two kinds of effects: intellectual deficit, which may be both general and specific, and personality manifestations. While this arbitrary dichotomy may disappear once the behavioral scientists begin to define intelligence more broadly (and realistically) (Haywood, 1967a), it is useful at present because it implies that both kinds of diagnostic procedures can be expected to yield data which bear upon the integrity of the central nervous system. Intellectual or ability deficits which frequently accompany cerebral dysfunction include impairments in speech and other language functions, both receptive and expressive, visual spatial disorders, disturbance of concept formation, sensory deficits and suppressions, and motor deficits, including pareses and plegias. Personality disorders accompanying brain lesions often mimic functional (i.e., nonorganic) personality disturbances and, particularly in children, yield such symptoms as unpredictable personality change, hyperactivity, depression, distractibility, and erratic aggressivity. Some investigators, notably Piotrowski (1957), have distinguished far more subtle personality correlates of brain disorder. Both intellectual and personality deficits will be treated in much greater detail in later papers in this series.

Psychometric Approaches to Neuropsychological Diagnosis

Psychometric instruments used for the assessment of brain damage can be categorized according to their usefulness in three functions: gross screening, lateralization, and localization. Gross screening refers to the process of making inferences regarding merely the presence or absence of encephalopathy. Lateralization refers to the process of determining whether a suspected brain lesion is located on the right or left sides of the brain. Localization refers to the process of determining the more precise locus of a focal lesion within one of the cerebral hemispheres. Other inferences can be drawn from psychometric data—for example, inferences regarding the etiology, extent, and velocity of a suspected lesion—but these are made primarily on the basis of particular patterns among scores on several instruments, usually taken together with social history data.

In the usual clinical neuropsychological examination, the order of investigation is the same as the listing here, i.e., the first step is a gross examination to yield inferences regarding merely the presence or absence of detectable encephalopathy, followed, if positive, by diagnostic techniques that will yield information regarding laterality and localization. It is the complex interactions among these successive steps in the neuropsychological examination that yield data bearing upon the finer inferences.

The early psychometric devices designed for detection of brain damage were mostly of the first type, i.e., brain damage was seen as a single entity which would yield reasonably uniform symptomatic characteristics, and the goal was simply to distinguish brain damaged from nonbrain damaged persons. The most successful of these procedures test functions involving perceptual motor or visual spatial operations, frequently through the copying of geometric forms. Such tests include the Bender Visual-Motor Gestalt Test, the Benton Visual Retention Test, the Graham-Kendall Memory-for-Designs Test, and sometimes tests involving the drawing of the human figure. Other reasonably successful devices designed to distinguish brain damaged from nonbrain damaged persons, but which do not involve the copying of geometric figures, include the Weigl-Goldstein-Scheerer series of tests (e.g., color form sorting), tests of concept formation by Vigotsky, the Shipley-Hartford Retreat Scale, and Reitan's Trail Making Test. While Halstead's Impairment Index is a much more complex procedure than any one of the foregoing, the gross score on the Impairment Index should be classed with these gross screening devices, since the inference commonly derived from the Impairment Index (Reitan, 1955a) alone has to do simply with the presence or absence of brain damage, even though much finer discriminations are made on the basis of the complex interactions among the components of the Impairment Index. Of these component tests, the most successful single instrument for revealing the presence of brain lesions in adult patients is the Category Test.

All of these procedures have had some success, in a statistical sense, in differentiating between groups of clearly brain damaged patients and groups of persons who have been either randomly selected or who have been judged by clinical experts to be free of any significant degree of brain damage. On the other hand, the very nature of the validation process for these instruments suggests that merely differentiating statistically between brain damaged and nonbrain damaged persons is no overwhelmingly beneficial accomplishment. Since the criterion procedures have already been able to do so, little is added by discovering that certain psychometric instruments show mean differences between these groups at the .05 or even the .001 levels. In other words, the clear differentiation of mean values of clinical groups is quite a different thing from the making of useful diagnostic and prognostic statements in the single individual.

In the gross screening lateralization sequence, each successive step has also the characteristics of the preceding step. Hence, procedures which are used to make inferences regarding the side of the brain on which a suspected brain lesion is located also obviously yield inferences regarding the presence or absence of such a lesion. Lateralization procedures are divided into two general categories: verbal nonverbal differences in intellectual performance and the contralateral mediation inference as applied to both sensory and motor aspects of performance.

It is by now fairly clear that the mediation of language skills occurs almost universally in the left cerebral hemisphere, and this is usually true even in left handed persons. Factor analyses of the Wechsler intelligence tests have upheld the general dichotomy of the subscales into verbal factors and performance factors (Cohen, 1959). Reitan and his associates have shown repeatedly that patients who have focal lesions of the left cerebral hemisphere usually have lower verbal than performance scores on the Wechsler-Bellevue Intelligence Scale, while patients with focal right hemisphere lesions have lower performance than verbal scores (Reitan, 1955b; Reed and Reitan, 1963). Hence, a very valuable assumption in the neuropsychological examination is that those functions which are tested by the verbal portion of the Wechsler intelligence tests appear to be mediated primarily in the left cerebral hemisphere, and those functions tested by the performance tests appear to be mediated primarily in the right cerebral hemisphere.

In spite of the apparent neatness of this arrangement, inferences based on this dual assumption need to take into account the absolute level of the full scale IQ in any given individual patient. The normal expectation with regard to verbal/performance discrepancies is that as full scale IQ deviates farther in a positive direction from the population mean (i.e., IQ 100), verbal score will ordinarily exceed performance score by an increasing amount, while at below average IQ levels performance IQ ordinarily exceeds verbal IQ, at least down to about IQ 50. Thus, if there is other convincing and independent evidence pointing to the presence of a brain lesion, the discrepancy between verbal and performance scores on the Wechsler intelligence scales can be useful in making inferences regarding the right left location of the suspected lesion. These tests are particularly useful, since within the verbal and performance categories they yield a further breakdown of abilities from which one can construct a more definitive profile of a patient's intellectual strengths and weaknesses.

One other verbal performance distinction has been shown to be quite useful in establishing the laterality of focal lesions. This distinction arises from the observation that patients with focal lesions of the right cerebral hemisphere do quite poorly at copying geometric figures, in particular a square, a triangle, and a Greek cross, especially when required to do so without lifting the pencil from the paper, but these individuals do not have correspondingly poor handwriting. Patients with focal lesions confined to the left cerebral hemisphere frequently show deterioration in handwriting, but they do not have comparable deficits in copying geometric figures (Heimburger and Reitan, 1961; Kløve and Reitan, 1958; Wheeler and Reitan, 1962). Thus, it appears that a reasonably intact left cerebral hemisphere is necessary for the adequate expression of language through handwriting, while the somewhat less complex drawing task involved in copying geometric figures from models appears to be mediated primarily in the right cerebral hemisphere.

A very useful piece of information in establishing laterality is that peripheral functions, both sensory and motor, have their central mediation on the side of the brain which is contralateral to the peripheral locus. Thus, an appropriately placed lesion of the right cerebral hemisphere may result in relative weakness and/or insensitivity on the left side of the body, while a similar left hemisphere lesion may produce comparable deficits on the right side of the body. Tests for lateral sensory functions include fairly standard neurological techniques for assessing tactile, visual, and auditory sensitivity, with the most useful data deriving from the patient's failure to report stimulation delivered to one side or the other under conditions of bilateral simultaneous stimulation (sensory suppressions). Useful tests for lateral differences in motor functions include finger tapping rate and strength of grip measurement (made with a good hand dynamometer).

Because of the exceedingly complex interacting communication network which constitutes the cerebral hemispheres, it is reasonable to expect that interruption of this network within the left cerebral hemisphere, even though the primary language centers are not directly involved, would impair the efficiency of language and of other left hemisphere functions to some degree. By the same reasoning, one would expect a lesion anywhere in the right cerebral hemisphere to impair the efficiency of essentially right hemisphere functions such as are measured by the performance tests of the Wechsler scales and by drawing tasks. In clinical neuropsychological diagnosis, it is usually the within individual comparison of these abilities that is of overriding importance.

The business of localization within the hemispheres is almost overwhelmingly complex. Except for a very few gross generalizations which are now possible, such localization has little clinical value in the individual case except for focal lesions which may be progressive, as in the case of cerebral neoplasms. The incidence of neoplastic disease in children, while certainly not negligible, constitutes a very minor portion of cases seen in child clinics. Because of this, and because the data are not nearly so clear as they are with respect to gross screening and lateralization, it may be sufficient to remind you here of two simple neuroanatomic observations.

The mediation of many important peripheral motor functions occurs just anterior to the fissure of Rolando, in the most posterior portions of the frontal lobes. This narrow anatomic region is referred to as the motor strip. There is a corresponding strip just posterior to the fissure of Rolando, along the most anterior portion of the parietal lobes, which mediates important peripheral sensory functions, and is known as the sensory strip.

It is clear that space occupying lesions in the frontal lobes will exert relatively more pressure upon the motor strip than upon its sensory counterpart across the fissure of Rolando, and that space occupying lesions lying posterior to the fissure of Rolando will exert relatively more pressure upon the sensory strip. Hence, space occupying lesions in the anterior part of the brain will impair motor functions relatively more than they will impair sensory functions, while space occupying lesions in the posterior portion of the brain will result in relatively more impaired sensory functions. From these characteristics of the effects of focal space occupying lesions, and from the general anatomic characteristics of the two halves of the brain divided by a coronal plain at the fissure of Rolando, it is grossly helpful to assume that anterior lesions will result in relatively impaired expressive abilities, while posterior lesions will result in relatively impaired receptive abilities. The understanding of these relationships will be considerably aided by a close examination of Netter (1962).

This gross distinction, even though less precise than one might wish, can be especially helpful in the neuropsychological diagnosis of children because of its obvious implications for educational planning. Even within the language area, it is possible that tests of receptive language, for example, the Peabody Picture Vocabulary Test, and other tests primarily of receptive language, may be usefully compared with tests of expressive language (for example, the oral vocabulary subtest on the Wechsler scales and the Stanford-Binet). Such intra-individual comparisons should yield profitable inferences regarding not only the relative integrity of anterior and posterior portions of the brain, but perhaps more importantly from an educational standpoint, inferences regarding relative strengths and weaknesses in expressive and receptive language functions in individual students for the purpose of planning the most helpful individual educational programs. Although it is still in the experimental stage, the Illinois Test of Psycholinguistic Abilities promises to be quite useful in this respect, since it yields scores on both expressive and receptive language functions. One would certainly hope for research with this instrument comparing the performances of children of varying age and IQ levels who have known anterior and posterior lesions, especially of the left cerebral hemisphere.

A final word on psychometric diagnosis is in order. Just as some brain lesions are neurologically silent, some are also psychologically silent, i.e., they may not yield the usual behavioral signs. For this reason, and also because it is logically impossible to prove the *nonexistence* of anything, the notion that one can rule out brain damage is patently ridiculous. The strongest negative statement a diagnostician can legitimately make is that his procedures have failed to find convincing evidence for the presence of a brain lesion.

The Meaning and Usefulness of Neuropsychological Diagnosis

The goals of neuropsychological diagnosis in children are many and may be as varied as the individual children who are brought into clinics. Among these various goals are three which have some degree of universality, and these will be discussed here.

From the standpoint of the scientific enterprise, it is quite important to be able to discover pathological organic states, and to relate specific pathological conditions to specific behavioral syndromes. Thus, we can gain some degree of understanding of the extent of correspondence between the condition of the brain and individual behavior. Such a contribution to the scientific enterprise is exactly what has motivated the largest efforts in clinical neuropsychology, notably those of Ward Halstead and Ralph M. Reitan. It is these same efforts, initially focused upon an effort to contribute to the fund of knowledge regarding brain behavior relationships, that have contributed some of the most useful clinical instruments for service use. Thus, the area of clinical neuropsychology, which cannot be truly experimental because of its necessary reliance upon clinical pathological material, provides a prime example of the close interaction between basic and applied considerations in science.

Another goal which is especially important, not because of its frequency, but because of the extremely undesirable consequences of inaction, is that of diagnosing progressive conditions for possible medical intervention. While progressive brain disease is relatively rare in children, as compared with its frequency in the adult population, (and especially as compared with the frequency of old, static, and diffuse encephalopathy in children), it is important to have instruments in one's psychometric armamentarium which are capable of diagnosing such conditions accurately. Most such conditions, if undiagnosed and untreated, will be much less treatable by the time they are apparent in everyday behavior, or may even result in death.

Perhaps the most important goal for clinical neuropsychology with children, especially in terms of the frequency of its applicability, is the goal of constructing profiles of abilities and deficits to make possible a more realistic educational plan. Haywood (1967b) has argued that in the case of school age children no diagnosis regarding brain damage should be rendered

> unless the following procedures are employed: assessment of intellectual ability using both verbal and nonverbal tasks; assessment of social functioning using something like the Vineland Social Maturity Scale; assessment of neuropsychological functions, using something like the Halstead-Reitan neuropsychological battery, the Bender Visual Motor Gestalt Test, the Benton Visual Retention Test, the Graham-Kendall Memory-for-Designs Test, and other neuropsychological procedures that have clear standardization norms; further assessment of perceptual functioning, using instruments such as the Kephart tests, the Frostig tests, and consultation with a neurologist and/or other specialist in the particular area of functioning which is thought to be deficient; educational assessment using standardized tests of educational achievement, and in particular using such diagnostic reading tests as the Durrell Analysis of Reading Difficulty and the Triggs Reading Survey; sensory

examination to test for deficiencies in vision and hearing; personality assessment involving the use of clinical interviews and standardized instruments for personality diagnosis. With such an exhaustive diagnostic survey, particular strengths and weaknesses, both in basic abilities and in learning achievement, can be spotted and a profile of these strengths and weaknesses constructed. I should emphasize particularly the need to chart a child's strong areas as well as his weak ones, since most diagnostic studies concentrate unduly on the latter. The construction of a useful program of instruction requires knowledge of strong areas of ability as well as weak ones, so that the strong areas can be used to stimulate work in the weak areas [pp. 11-12].

Of the many significant questions to be asked regarding the usefulness of clinical neuropsychological diagnosis in children, the following seem to commend themselves especially to close scrutiny and possible research:

1. Can the locus, extent, and probable etiology of a suspected lesion be established with psychometric techniques?
2. Does a diagnosis of locus, extent, and probable etiology carry specific implications for educational treatment?
3. Can specific training procedures alter the behavioral condition?

Some suggestions pointing toward possible answers to the first question will surely be gained from Dr. Reed's paper in this series. The success of such investigative programs as those of Halstead and Reitan and their associates is clear. These programs have investigated behavioral effects of focal lesions in the human adult, and have done so primarily in a closed population. Most of the patients who have come to their attention may reasonably be assumed to have had at one time a fully developed and adequately functioning nervous system. To establish similar relationships in children, whose nervous systems are still undergoing considerable development, appears to be a much more complex problem. In undertaking this task, one should certainly be encouraged by the noteworthy successes of programs of neuropsychological investigation with adults, but one should also understand that the number of interacting variables will be somewhat greater. In adults, the interacting variables include at least the following: locus, extent, etiology, velocity, and premorbid level of functioning. When working with children, one should add to these at least the following: age at onset, chronicity, and for prognostic inferences, present chronological and mental age and availability of remedial procedures.

Much of this difficulty arises from the established observation that the human nervous system is far from fully developed at birth, and continues to develop at a progressively decelerated pace for at least the next twelve years. Thus, there is greater opportunity for compensatory functioning if damage to the nervous system occurs during the developmental period. This is not to say that any brain lesion occurring at any time during development necessarily has a better prognosis for recovery of function than if the same lesion should occur in the fully developed brain. If there is accuracy in the critical periods hypothesis with respect to the development of neural structures, one would expect an interaction to occur between the location of a brain lesion and the stage of development during which it occurs. Thus, as in any other clinical investigative process, a most careful developmental history will be of great value.

In addition to the usual milestones history, the clinician will need to have very specific information regarding the following points: (a) any injury to the head, including the precise location, whether the child lost consciousness and for how long, and whether medical attention was available; (b) the child's behavior both immediately and for several days following the injury, and whether or not there was a noticeable personality change; (c) any occasions of ingestion of toxic substances, and the sequelae; (d) occasions of prolonged nausea and vomiting, which could not be related to eating habits or severe emotional upset; (e) periods of very high body temperature, including if possible the precise temperatures reached and the duration of these, as well as whether there were febrile convulsions; and (f) any sudden changes in levels of energy expenditure.

In spite of the overwhelming complexity of establishing brain behavior relationships in children, some investigators have begun to have some success in this area. Particularly notable among these are Frances Graham of the University of Wisconsin, and Homer B. C. Reed, Jr., from whom you will get more specific information in the present series.

Two further problems inevitably arise to plague the practice of clinical neuropsychology in a population of children. The more glaring of these problems is the present inadequacy of normative data on the development of neuropsychological relationships by age and intelligence levels. The other is the problem of minimal brain damage, about which there will be a great deal more in Lloyd Dunn's paper.

In clinical neuropsychological investigation with adult subjects, the acquisition of normative data for one's techniques is made difficult primarily by the investigator's dependence upon clinical pathological material. Thus, while it may take many years to accumulate an adequate group of patients who have similarly placed lesions of similar etiology, the problem of age norms is not crucial, and very gross age categories will suffice. This is true because most investigators in adult clinical neuropsychology have dealt primarily with acute brain lesions, or at least have been able usually to assume that their patients have at one time had a fully developed and reasonably intact nervous system.

The problem of normative data developed by much finer age categories is much more crucial in the case of children. A chief reason for this is that many neuropsychological diagnostic procedures are closely related to tests of ability or intelligence, and such abilities obviously increase with increasing chronological age, reaching a near asymptote in late adolescence or early adulthood. Because of the moderately strong correlations between IQ scores and scores on many neuropsychological diagnostic instruments, if one established cutting scores on the tests on the basis of examination of twelve year old children, it is likely that most six year old children would be classified as brain damaged.

Another deterring factor is related to the observation that child clinics are filled with children who are thought to be brain damaged on the basis of erratic social behavior and scholastic performance, but whose brain damage cannot easily be related to any specifiable etiology. Such children may never have had a fully developed and intact nervous system, since their deficiencies might have resulted from genetic, intrauterine, or perinatal influences. Thus, it is imperative that the most promising neuropsychological diagnostic procedures be subjected to the most careful standardization, including samples at frequent chronological age levels, and within each level several categories of psychometric intelligence. For such a long and arduous standardization effort, it will be necessary that there be some central collection and data processing agency, such as the Southern Regional Education Board might represent. The task is within the realm of possibility if those clinicians who actually see such children in diagnostic centers would accumulate case records, complete with social histories, descriptions of present functioning, and comprehensive test data, for periodic forwarding to the central collection and data processing agency.

The problem of minimal brain dysfunction is especially relevant to diagnostic work with children. In the present author's opinion, the primary goal of diagnostic work with such children is not labeling, or even intervention for the sake of forestalling progressive disease, but is rather the construction of adequate individual programs of education and training which will depend upon most complete knowledge of each child's profile of strengths and weaknesses across many areas of functioning. The day is now long past when a general estimate of overall intellectual functioning was thought to be especially helpful in educational planning. It is increasingly apparent that there is not an intelligence, but instead there are many intelligences, and that the profiles of these different areas of intellectual functioning vary considerably from child to child. The mere recognition that a child's difficulty in reading, for example, is related to a specific brain lesion rather than primarily to his intransigence or more generalized mental retardation, is in itself educationally helpful (Reitan, 1966). Beyond that consideration, a broad profile of individual strengths and weaknesses can help the educator to devise programs based upon different areas of ability. Whether the educator adheres to the teach to strengths school or the teach to weaknesses school, he must know first what the areas of strength and weakness are, and, if possible, whether the weaknesses have a structural basis.

Who Should Diagnose?

Questions frequently arise regarding the appropriateness of diagnostic statements about brain damage coming from different disciplines. The question is, simply, Who should offer diagnostic opinions? Even in the case of acute and/or progressive brain lesions in which it is quite clear that the physician must have ultimate medical and legal responsibility for the care of the patient, it is legitimate to ask what disciplines should participate in the diagnostic process, and how loudly and insistently each diagnostician may offer his opinion. The answer would seem to be that nonmedical persons should participate to the extent to which their own training and experience permit them to make accurate diagnostic inferences. The physician charged with the care of the patients in whom brain lesions are suspected cannot afford to ignore potentially useful information from whatever source it may come. Haywood and Hamlin (1963) have defined the issue in terms of the consequences of being wrong versus the consequences of being right, when nonmedical practitioners make diagnostic inferences particularly with respect to suspected progressive lesions. If such a person insists that the patient has a progressive brain lesion when in fact he does not, the usual result is some professional embarrassment for the practitioner and possibly some inconvenience and needless expense for the patient. On the other hand, if such a practitioner suspects the presence of a progressive lesion, is correct in his assumption, and remains silent rather than insisting upon further examination, he will have shown himself to be of very limited usefulness in his clinical setting, and the result for the patient may very well be death. Seen in these terms, if there must be diagnostic errors in the case of progressive lesions, there can be little doubt as to the more desirable direction for the errors to take.

As usual, the rather extreme case has been drawn most sharply, even though it does not represent the largest number of neuropsychological investigations encountered in child clinics. In the more usual situation, one is dealing with old and static lesions which are not sharply focused, and whose behavioral effects are diffuse. In such cases, medical collaboration is desirable both because of the possibility of chemical control of symptoms, and because the particular nonmedical practitioner may not have the most complete picture of the deficits involved. Such a statement neither requires nor implies a need for medical direction of psychological and educational diagnostic studies. The psychological or educational practitioner should assure himself that those who receive his attentions, whether or not he suspects the presence of brain lesions, have adequate medical attention (Pennington and Berg, 1954). Proceeding from that point, the most appropriate person to offer diagnostic opinions is the one whose instruments can measure variations in individual performance across a wide range of abilities and functions, and who has sufficient acquaintance with the education of children to be able to make useful recommendations regarding individually constructed educational and training programs. The conclusion is that the neuropsychologist who wishes to practice with children should acquire not only a thorough knowledge of functional neuroanatomy, neurology, and psychometrics, but should also acquaint himself with available educational procedures and the relationships between differential abilities and deficits on the one hand, and the requirements of such educational procedures on the other. It will be immediately obvious that such persons are unfortunately rare. The equally obvious alternative is for the neuropsychologist to ally himself with appropriately trained physicians and educators.

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Part 2:

Neurological Foundations and Related Problems

An Overview of the Organization of the Central Nervous System

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By way of a preface to this chapter, it should be pointed out that in any discussion of neuroanatomy the reader is overwhelmed with numerous terms; specifically, those which are used to identify specific structures within the brain and those which identify the spatial relationships between structures. Most of the material in this chapter will serve as an introduction to some of the specific structures within the brain, but to facilitate this discussion it will first be necessary to define the terms which describe the interrelationships between these structures.

All mammals have a midline about which they are symmetrical. The terms medial and lateral describe the location of structures that are toward or away from that midline, respectively. Similarly, the term dorsal is used to refer to structures which lie in the direction of the back or top of the animal. Structures which are ventral lie in the direction of the abdominal side of the animal. All mammals have an anterior and a posterior end; those structures lying toward the anterior or front end are cranial or rostral to structures lying in the posterior or caudal direction.

The rest of the material included in this chapter will give the readers some familiarity with the most frequently used neuroanatomical terms. The section on the growth of the brain includes many terms which are derived from a consideration of the embryological origin of specific neuroanatomical regions. The sections on the integrative aspect of the brain include a description of many of the neuroanatomical structures that can be delineated *within the brain.*

The reader interested in a more detailed knowledge of neuroanatomy than will be presented here is referred to one of the many excellent texts on the subject, such as Ranson and Clark (1959), Elliott (1963), Peele (1961), or Arey (1954).

The assessment of damage to the central nervous system must necessarily be preceded by a knowledge of the functional parts of this highly complicated structure. There must also be an awareness of the relationship between the various parts of the central nervous system and the observable dimensions of their action, i.e., the behavior of the organism.

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However, the extent of this knowledge is limited by the progress of the particular sciences which have addressed themselves to these problems. In recent years, the science of neuropsychology has made great strides in understanding the manner in which brain mechanisms regulate behavior. Yet, at best, the extent of our understanding of the anatomical behavioral interrelationships has only just begun.

The material presented in this chapter will attempt to examine some of the interrelationships between the brain and behavior within the context of the growing organism. Our purpose is fourfold: (a) we will examine the origin of some gross neuroanatomical structures within the context of their development; (b) we will attempt to suggest some generalities about the manner by which insult to the developing brain might alter brain behavior relationships; (c) we will review some of the functional attributes of neural structures in terms of the current research on brain behavior relationships; and (d) we will briefly examine some of the interactions between the organism's experiences during development and damage to the central nervous system. It is the intention of the authors that the suggested relationships might then serve as guides to the practitioner concerned with the assessment of brain damage in the young and as hypotheses to the research worker concerned with the investigation of the developing brain.

The Growth of the Brain

Embryological Development. The development of the nervous system begins with the formation of a neural tube from the outside cell layer of the embryo. The nerve cells in this tube soon send out small extensions called axons, which subsequently connect adjacent parts of the neural tube. It is the axon which gives the nervous system the capacity to send information from one part of the body to another. Thus, the axon serves as a conductor in much the same sense that an electric wire conducts. At very early stages in the growth of the organism the developing nerve fibers or axons can be separated into afferent (input) and efferent (output) nerves on the basis of their position within the neural tube. The axonal processes which lie in the ventral or bottom portion of the neural crest become the efferent fibers which ultimately connect the central nervous system with the muscles and internal organs of the body. The dorsal nerve fibers, those on the top of the tube, are afferent and relay neural information from the periphery to the central nervous system. These nerves conduct sensory information into the central nervous system.

The greatest changes in the structure of the brain are taking place in the gross development of the brain during the period from the fourth week to the fourteenth week of gestation. At the beginning of this period the cephalic (Gr: kephale, head) end of the neural tube is characterized by three indentations: the prosencephalic vesicle, the mesencephalic vesicle, and the rhombencephalic vesicle. As nerve cells multiply, the region of the brain elongates and bends in upon itself as shown in Figure 1.

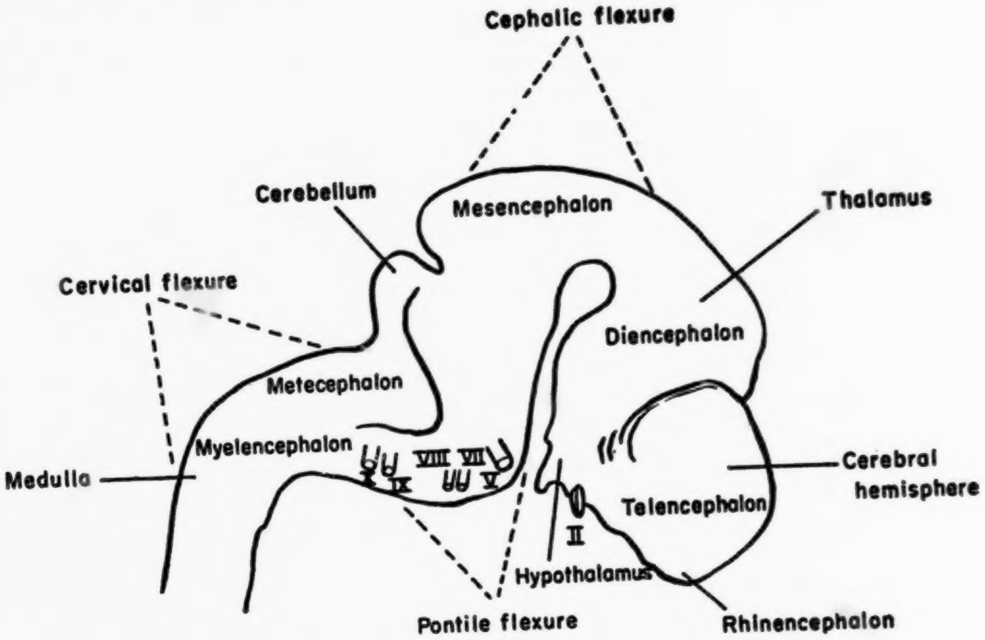
Cephalic flexure
Cerebellum
Mesencephalon
Thalamus
Cervical flexure
Diencephalon
Metecephalon
Myelencephalon
VIII
VII
Medulla
X
IX
V
Cerebral hemisphere
Telencephalon
II
Hypothalamus
Pontile flexure
Rhinencephalon

FIGURE 1. *The brain of an embryo at approximately six weeks of gestation. The cerebellum, thalamus, hypothalamus, and cerebral hemispheres develop from the embryological divisions depicted in the figure. The Roman numerals refer to the numbers of the cranial nerves illustrated in the drawing (Ranson and Clark, 1959).*

By the sixth week of gestation the major embryological divisions of the brain are discernible. The anatomical terms which are used to describe the embryological divisions are used repeatedly in the description of the brain, so their origin is worthy of some elaboration. The most posterior lobe immediately anterior to the spinal cord is the rhombencephalon which is divided roughly in half by the pontile flexure. Posterior to the flexure is the myelencephalon (Gr: myelos, marrow), an area which ultimately develops into the medulla oblongata. Anterior to the flexure, on the dorsal surface of the brain, is the metencephalon (Gr: meta, between). The cerebellum and the pons take their origin from the metencephalon.

The midbrain or mesencephalon (Gr: mesos, middle) is an elaboration of the mesencephalic vesicle and the curvature of the cephalic flexure. It is bounded on the under surface by a tegmental (L: tegmina, cover) layer of cells. In addition to being the origin of a number of cranial nerves, the mesencephalon is an area of numerous fiber tracts which interconnect the posterior and anterior neural structures.

The prosencephalon (Gr: proso, before), which develops around the prosencephalic vesicle, is typically divided into two subsections: the diencephalon (Gr: dia, through) and the telencephalon (Gr: telos, end). These two subsections are separated by a constriction within the internal portion of the prosencephalic vesicle. The diencephalon includes the cells which ultimately make up the areas of the thalamus, the epithalamus, the metathalamus, the hypothalamus, and the subthalamus.

The telencephalon is particularly important within the context of human development, since it is from this structure that the great cerebral hemispheres originate. In addition, the telencephalon includes a number of structures which are located within the cerebral hemispheres, such as the hippocampus, the pyriform cortex, the entrorhinal cortex, the amygdaloid complex, the corpus callosum, and the basal ganglia. With the exception of the latter two, these internal structures are called the archipallium (Gr: arche, beginning, and L: pallium, a cloak) since they are phylogenetically older cortical structures which are highly developed in submammalian orders such as reptiles, as well as in early mammals. The neopallium (Gr: neos, new), which is a relatively recent phylogenetic structure, has evolved to be man's most distinguishing characteristic, the neocortex.

In general, the gross structure of the brain has reached its adult form by the fourteenth week of gestation, but the development of the brain is far from complete by this age. The neocortex has not yet begun to invaginate, or enfold upon itself, and only a relatively few of the billions of cells which characterize the adult brain are present. However, this embryological stage of development is important within the context of this volume for two related reasons. First, the embryological period represents a critical time during an organism's growth because it marks the developmental phase in which the gross structural relationships within the brain are ordered. Secondly, this period represents a time in development when the brain is maximally sensitive to alterations by environmental insult. Considering the immensity of neural development which takes place as the embryo is growing, it is not difficult to visualize how susceptible the system would be to morphological distortion should any one structure malform. Consequently, central nervous system abnormalities which occur during this period are usually accompanied by gross structural abnormalities which aid considerably in the diagnosis of brain damage.

Foetal Development. The brain continues to grow from the twelfth week of gestation on into adolescence. Enfolding of the neural tube is completed by 14 weeks, but much more enfolding and invagination of particular structures will take place before birth. The foetal period from about 12 weeks after gestation until birth is characterized by the elaboration and extension of the basic arrangement of the cells that were laid down in the embryo. On the cellular level, growth during this period is characterized by cellular movement and differentiation. These changes lead ultimately to the extensive interconnections among nerve cells which permit interactions between neural structures.

This is an extremely important period of growth. Although the axons can carry information from one place to another, nerve cells are physically separated from one another. This separation is called a synaptic cleft and the region in which two nerve fibers come into proximity is called a synapse. It is at the synapse that the exchange of information between nerve cells occurs. This process of information exchange is the most important function of the nervous system, for it represents the fundamental integrating operation of the nervous system. The nervous system is essentially nonfunctional until the synapse becomes an operational unit. Cellular differentiation and the growth of the extensions of the cell body bring nerves into proximity and allow information exchange between cells to occur.

It is probable that damage to the brain during this period of growth will result in functional anomalies that are not necessarily correlated with observable gross structural anomalies. Such damage may serve to arrest the development of cells of the brain and, as a result, prevent the final elaboration of the normal cellular arrangements. Such insult would probably result in severe retardation of the functional expression of the brain's activity. The cerebral cortex is particularly sensitive to abnormalities of this sort since it lies on the outside of the developing nervous system.

Postnatal Growth. The brain of the newborn is far from completely developed, and it is possible to discuss in some detail the several dimensions of postnatal growth that have been assessed fairly extensively. Much of this work has been done on experimental animals. In terms of the growth of the brain, there is a high correlation between human and subhuman neural development, i.e., both show similar patterns of development in the brain.

Morphological Growth. The appearance of the infant brain on a gross anatomical level is much the same as a mature brain. The fissures or indentations in the cerebellar and cerebral hemispheres, which begin to appear at about 24 weeks of gestation (Dodgson, 1962), are basically completed at birth. Although the total weight of the infant brain is only about 25 percent of that of the adult, it is still possible to recognize all the structures that are present at maturity. On the microscopic level, there are still two major patterns of development that must occur: (a) the formation of the myelin which covers the axons, and (b) the elaboration of the dendrites of cells.

Cellular Growth. The formation of myelin is a major factor contributing to the increase in brain weight from infancy to adulthood (Davison and Dobbing, 1966). Myelin is a multilayered fatty tissue which covers certain nerve fibers from the body of the cell to the vicinity of the nerve terminal. This coating, or shielding, apparently serves two functions for the nerve: it protects and supports the axon, and it functions to increase the efficiency of neural conduction. Experimental evidence indicates that nerve fibers that do not have a myelin covering in the newborn show a high rate of fatigue, i.e., they do not recover from a series of neural impulses as rapidly as do myelinated fibers in older animals. This observation indicates that myelination does result in an increased efficiency of neural transmission. For this reason, the pattern of the development of myelin can be taken as an index of the functional development of different parts of the brain.

Within the cerebral cortex, there appear to be differential patterns of growth. Myelin begins to develop first in the areas which are related functionally to the somesthetic senses and motor activity. This is followed by myelination in the visual and auditory areas and finally by myelination of the association areas of the cortex (Dodgson, 1962). In humans myelination occurs most rapidly during late prenatal and early postnatal life, though the process continues on into adolescence.

The elaboration of the dendritic processes of nerve cells is the second substantial factor in postnatal brain development. The dendrites are extensions of the cell body and they bring nerves in closer proximity to axons, thus facilitating information exchange. Before the neural systems which control behavior can achieve functional integrity, there must be a system of neural connections. The axons, and particularly the finely aborized dendrites, provide the neural telemetry by which brain components become related and brain mechanisms become functional.

Though there is an increase in the size of the cells in some areas of the brain following birth, there is little dendritic elaboration before two years of age. Thereafter the dendrites continue to grow until maturity.

It is reasonable to suggest that much of the complex behavior that appears in the developing organism occurs concomitantly with the growth and connection, via the dendrites, of the neural circuits that regulate behavior.

Insult to the newborn organism which affects the growth of the brain generally represents insult to the developing cells of the brain. Since the cells which regulate the elaboration of functional capacities that are exhibited by the organism grow at different rates and at somewhat different times during development, the age at which the insult occurs may have its effect on different functional systems of the brain. For this reason, it is difficult to be definitive in qualifying the nature of behavioral disruption which ensues from postnatal trauma to the central nervous system. A general guide might be that the trauma would result in an eventual deficit in the infant for integrating neural information. The deficit would, or course, be related to incompletely or inappropriately developed systems of neural connection, i.e., poor dendritic or axonal elaboration.

Biochemical Growth. Just as it is obvious that an organism lacks certain functional capacities of behavior until such time as the appropriate anatomical structures are complete, it is also obvious that functional capacity is dependent on appropriate biochemical development. In a very real sense, any deficit in the behavior of an immature brain can be related to the biological integrity of the biochemical systems of the brain.

The neural transmitter substances are an important class of brain chemicals to be considered because of their implicated function in the regulation of behavior. These chemical substances bridge the structural gap between nerve cells at the synaptic cleft and thus mediate the transport of neural information at the synapse. Several chemical compounds, including acetylcholine, norepinephrine, and serotonin, have been suggested as possible neurohumors or neural transmitters in the human brain.

The ontological appearance of neurohumors and their related enzyme systems reflects the development of the "chemistry of function." The organism develops those chemical systems which will permit a sufficient degree of function to cope with an extrauterine environment. Since neurohumors are essential to neural functioning, it is appropriate that they should appear prenatally and expand considerably during postnatal development. Thus one may roughly equate neurohumoral development with early functional development.

Electrophysiological Growth. Perhaps one of the points of greatest fascination to the neurologist is the electrical behavior of the brain, especially that revealed by the electroencephalograph (EEG). Although electroencephalography does not present the investigator with a technique for the exploration of all parameters of brain activity, it does represent a sufficiently developed methodology for characterizing certain patterns of brain activity. The EEG of the mature organism may be differentiated into two primary waveforms: (a) alpha rhythm, which is an oscillation of 8 to 10 cycles per second and is of rather high amplitude; and (b) beta rhythm, which is from 25 to 50 cycles per second and is of lesser amplitude. Alpha rhythm is generally associated with a nonalert state in the organism. It is typically recorded when the brain is not involved in any mental activity and external stimuli are minimized by shutting the eyelids. On the other hand, when an individual engages in any sort of mental task, the beta rhythm predominates.

While it is possible to record the EEG of a foetus, activity is slow and quite unlike that at maturity. There is actually a third rhythm, the delta rhythm (1 to 5 cycles per second), which is characteristic of the immature brain and persists into adolescence, at which time the alpha and beta rhythms become more distinct. For this reason, the EEG has limited application to the assessment of brain damage in children.

In the resting adult, it is possible to elicit a disruption of the alpha pattern following both auditory and visual stimuli. At birth, the electrical activity of the infant brain responds to auditory stimuli in a diffuse and variable manner, but responsiveness to visual stimuli is distinct and always detectable (Ellingson, 1964). The changing character of the postnatal EEG may be an expression of the elaboration of the dendrites that are evolving in the cortex.

The evoked potential is also an important electrophysiological index of function. In the adult brain, stimulation of a peripheral receptor organ will produce a recordable electrical response in those parts of the brain to which the nerve cells of the receptor system project. For example, stimulating the eye with light will result in an electrical potential which lasts for about 80 msec. and can be recorded from the posterior lobe of the cortex. In the adult, this potential is characterized by an initial positive component of about 150 volts followed by a negative potential of similar magnitude. The maturation of this response has been studied by several investigators, and their results are generally in quite good agreement. In the young brain, the initial positive segment is virtually nonexistent. As the organism matures, the positive component of this potential increases. Pupura and his colleagues (Pupura, Shofer, Housepain, and Noback, 1904) have demonstrated that the development of the basal dendritic system of cortical cells correlates very highly with the development of the initial positive segment of this wave.

The latency of the evoked response also changes with age. In the human visual system there is a two to fourfold decrease in latency between birth and adulthood. Indeed, Ellingson (1960) has shown that there is an inverse correlation (r — .80) between age and latency of the evoked cortical response from birth to an age at which the body weight reaches about 12 pounds. This change in response latency may be related to myelination and biochemical development in the neural system that mediates the transmission of the evoked potential.

Functional Growth. Reflexive responses are the most widely investigated index of functional development, for it is the simple reflex that is the precursor to the elaboration of complex behavior. However, it is apparent that in even the simplest of reflexes there are many contributing components that must mature sufficiently for the reflexive pattern to appear. The receptor and effector organs must develop to receive and respond to their appropriate stimuli. The nerve cells must achieve some degree of cellular differentiation and development. Furthermore, there must be sufficient biochemical development to allow the nerve impulse to be transmitted at the appropriate synaptic junctures.

During embryological development, the central nervous system is an increasing mass of differentiating cells with little responsibility for functional mediation. As the foetus develops, however, an increasing number of reflexive behaviors are observable in the organism. This development is due primarily to the development of those elements of the reflex that allow an integrated pattern of behavior to occur. An excellent example of this structural functional interdependency can be observed in the development of the foetal grasp reflex. On the structural level, the nerve fibers and sensory and motor elements that mediate the reflexive pattern develop before the reflex. At about the sixth week of gestation, the ventral (motor) roots of the spinal cord grow outward from the cells of the anterior grey horn to make contact with the striate muscle fibers in the limb bulbs. The development of the dorsal (sensory) root ganglia, which lie outside the spinal cord, follows a pattern similar to the ventral roots, but they grow into the spinal cord to end in the region of the posterior columns. The development of the neural tracts, which allow communication within the spinal cord, follows the growth of the sensory and motor roots. Finally, the nerve bundles such as the spinothalmic tracts and the corticospinal tracts, which connect the spinal segments with higher brain centers, begin their development.

The response of the hand to tactile stimulation does not appear in the foetus until about the ninth week of gestation, or after the development of the sensory muscular neural apparatus. Prior to this time, cellular differentiation in the motor groups of the cervical spinal nerves innervating the hand is virtually nonexistent. From 11 to 28 weeks of menstrual age, the grasp reflex to tactile stimulation of the palm shows extremely rapid development as do the cells which innervate the flexor and extensor muscles of the fingers and hand. Though a weak grasp reflex is present at 18 weeks, it does not become an integrated response involving all parts of the hand until about 28 weeks. During this time period, the cells of the spinal cord which allow communication between spinal segments are rapidly developing. Thus the refinement of the reflex accompanies the development of those neural components which are essential for the reflex (Humphrey, 1964).

The postnatal development of the manipulative skills of the hand have been studied in detail by Halverson (1937a, 1937b, 1937c). A strong reflexive grasping response can be elicited at birth by stimulation of the palm, but between 24 and 52 weeks in the postnatal period, its strength diminishes rapidly, giving way to voluntary control of the hand and fingers. Voluntary reaching and grasping first appear at about 20 weeks, but the response is very primitive and involves the pressure of the fingers against the hand. As the infant matures, the efficiency, durability, and flexibility of grasping increase. By 32 weeks, an object is grasped by asserting the pressure of the fingers against the base of the thumb, and by 52 weeks, objects are picked up and held with the forefingers and the thumb.

It is probable that the use of the hand in voluntary grasping is correlated with the development of neural control systems of the hand and fingers which are organized in the central nervous system at levels higher than the spinal cord. Supraspinal reflexes are deficient at birth, but as development proceeds and higher levels of neural control become integrated with spinal control mechanisms, fine musculature control becomes possible. Morphological and biochemical development play a significant role in this process. The presence of myelin increases the functional efficiency of the responses. The interactions between the nerve fibers that innervate the muscles necessary to perform a coordinated response are greatly facilitated by an elaboration of the dendritic processes of these nerve cells. Biochemical development allows further motor control by introducing systems of simultaneous excitation and inhibition at the synapses, thus facilitating the discrete actions of particular muscle groups that must function when the response is made.

These data serve to demonstrate that anatomical functional interdependencies, as they appear during development, result in the regulation of a particular form of behavior. They further indicate that the patterns of neural development prescribe the order and complexity of the observable functional characteristics as they are manifested in the organism's behavioral repertoire. It is possible that similar temporal relationships could be observed between neural growth and the development of other forms of behavior of a more complex nature. Empirical substantiation of this hypothesis necessitates detailed observations on both behavioral development and anatomical development. Such correlations might prove to be extremely informative.

Organization of Functional Systems in the Brain

Having introduced the overall organization of the brain via its development, we now turn to an overview of some of the functional systems within the brain. Within this context, it is first necessary to state the assumptions which serve as guidelines for the material that will be covered.

An organism's reactions to environmental events are the fundamental observations of the behavioral sciences. This view assumes that all behaviors are elicited by the presence of some stimulus that exists either in the organism's internal or external environment. This behavior represents a series of reactions to stimuli that increase in their order of complexity from simple reflexes to integrated, organized patterns of motor behavior. Within this context, the function of the brain is to regulate the behavior of the organism in the presence of the myriad of stimuli that are imposed on it at any given time.

On the molecular level, the basic unit of the nervous system and thus of behavioral regulation is the nerve cell. This structure, by means of its axonal and dendritic extensions, provides the organism with a mechanism both for relaying information from one part of itself to another and for reacting to that information. The synapse, or the junction between neural units, gives the nervous system the capacity to modulate and modify the transmission of neural information. To make the statement more specific, consider a simple organism with a two unit neural system composed of one afferent and one efferent fiber and an interposed synapse. In such a system, given a knowledge of the state of excitement of the afferent fiber, one could predict with almost absolute certainty the action of the efferent fiber and the behavior of the organism.

In the mammalian nervous system, such a simple two unit system is the exception rather than the rule. Any one nerve cell is surrounded by many others which probably have a similar function. In addition, every cell has hundreds if not thousands of impinging synaptic junctions. When one considers further the probable fact that not all fibers are releasing the same neurohumor, and that each neurohumor produces its characteristic transmitting effect, the complexity of the nervous system is increased multifold. Furthermore, only certain synapses can excite nerve cells. Others inhibit neural transmission.

The high degree of complexity makes it virtually impossible to present a complete analysis of brain function, even if the state of the science were such that all the details were known. We will, therefore, be very selective in the material that will be reviewed below, and limit our presentations to only a relatively few of the functional systems of the brain.

Input and Output Systems. The spinal cord and its nerve fibers are very important in the majority of neural systems, since it is via these tracts that nerve impulses from the body reach the brain and vice versa. Sensory information from the periphery enters the spinal cord via the dorsal root. These nerves carry sensory input from the receptors which have appropriately encoded the environmental stimuli such as touch, pain, temperature, and proprioception. Following entry into the cord, the sensory systems are organized into neural tracts which lie on the dorsal external surfaces of the cord where they project upward into the brain.

Output to the muscles in the body proceeds from the spinal cord via the ventral root. There are two classes of motor neurons which leave the spinal cord via the motor roots: those which innervate the skeletal muscles and thus control the so called voluntary actions of the body, and those which innervate the smooth or involuntary muscles of the viscera. For the thoracic and lumbar segments of the spinal cord, this latter group of fibers form synapses outside the spinal cord in the sympathetic ganglia. These ganglia are clusters of nerve cells which extend from the base of the skull to the terminal region of the vertebral column as the sympathetic trunk. The fibers which arise from these ganglia (postganglionic efferents) ultimately project to the visceral organs of the gut. This system constitutes the sympathetic division of the peripheral nervous system, and Cannon and Rosenblueth (1937) have suggested that activity in the sympathetic nervous system prepares the body for overt action.

The parasympathetic nervous system constitutes another important effector system which innervates smooth muscle. It differs from the sympathetic system in several respects. Parasympathetic fibers arise from the cranial and sacral segments of the spinal cord and from some of the cranial nerves located within the brain. The parasympathetic ganglia, unlike the sympathetic ganglia, are commonly located very near the organs that the nerves innervate, where they synapse with relatively short postganglionic fibers. Cannon and Rosenblueth (1937) have proposed that this system regulates the recuperative processes of the visceral system and functions to establish and maintain balance in the visceral organs of the body.

A further distinction between the sympathetic and parasympathetic systems is typically made on the basis of the neural transmitter substance liberated at the postganglionic nerve terminals. In the sympathetic system, the majority of fibers liberate norepinepherine or noradrenalin, and this system is referred to as adrenergic. The parasympathetic postganglionic fibers release acetylcholine and are thus called cholinergic. The regulation of the internal visceral organs, so important in maintaining homeostatic equilibrium, thus can be viewed as a balance between adrenergic and cholinergic neural mechanisms.

Both the afferent and efferent fibers of the body are collected in one nerve bundle, called the spinal nerve, as they enter and leave the spinal cord. Spinal nerves are segmentally arranged so that each nerve supplies only a particular portion of the body surface. The area of the body innervated by a particular motor segment is called a myotome. The sensory fibers supply a body segment called a dermatome. Because of the high degree of organization of these segments within the cord, damage to this part of the nervous system is relatively easy to assess. The loss of pain and temperature sensitivity in a particular spinal segment of the body would most likely be produced by damage to the lateral spinothalamic tract. Damage to the ventral horn cells, such as that produced by poliomyelitis, results in the atrophy and paralysis of muscle groups innervated by those cells.

The spinal cord extends anteriorly to become the myelencephalon of the brain. The myelencephalon and the metencephalon, excluding the cerebellum, are often referred to as the brain stem because in lateral view they appear as a stem to the more anterior portions of the brain (see Figure 2). The more rostral parts of the mesencephalon may also be included in this term.

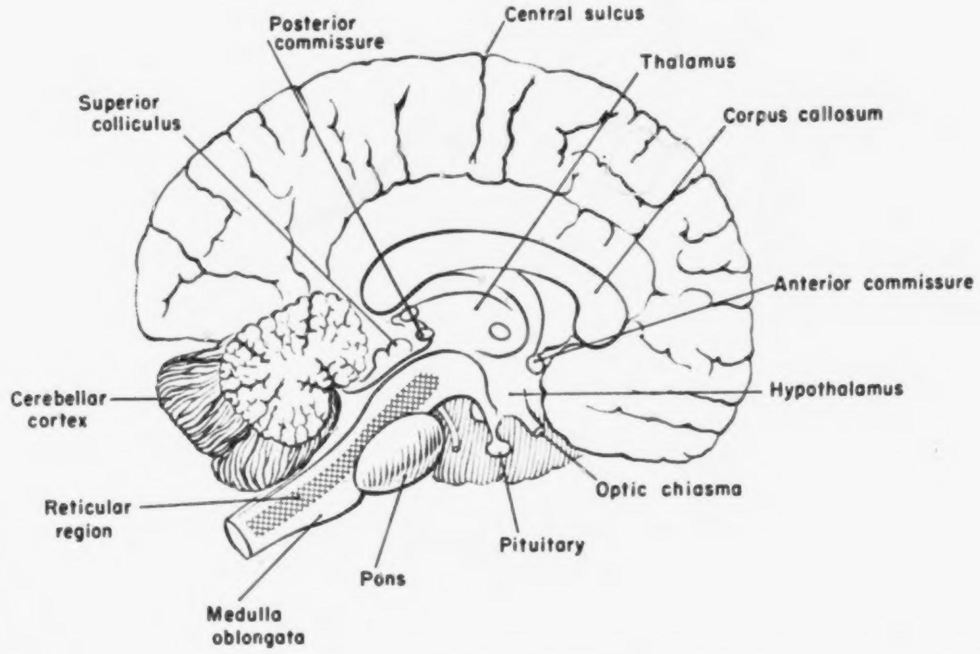
Posterior commissure
Central sulcus
Thalamus
Superior colliculus
Corpus callosum
Anterior commissure
Cerebellar cortex
Hypothalamus
Reticular region
Optic chiasma
Pituitary
Pons
Medulla oblongata

FIGURE 2. The midline of a brain showing some of the major gross structures mentioned in the text (Ranson and Clark, 1959).

The nuclei of ten of the twelve cranial nerves lie within the brain stem; these nerves represent the major input and output systems whose origins are located within the brain. The location and function of the cranial nerves are summarized in Table 1. Some of the cranial nerves have both a sensory and motor component; others have only a sensory or a motor function. Furthermore, the motor nerves may innervate either visceral or skeletal muscle. A thorough knowledge of the location and function of these nerves is an important aspect of the assessment of damage to the central nervous system, since these nerves represent the primary sensory and motor nuclei located within the brain.

TABLE 1

Cranial Nerves: Origin, Course, Termination, and Function

Cranial Nerve	*Origin and Course*	*Termination and Function*
1. Olfactory:	Nasal Mucosa. Passes through cribiform plate to olf. bulb then along olf. nerve below frontal lobe.	Medial branch projects to contralateral bulb. Lateral branch ends is pyriform and amygdala. Smell.
2. Optic:	Retinal ganglion cells. Hemidecussation of the nerve occurs at optic chiasm and projects to lateral geniculate body of thalamus.	Projections from the geniculate go to occipital cortex. Vision.
3. Oculomotor:	Oculomotor nuclei on the floor of the cerebral aqueduct. Emerges at upper pons and passes through lateral wall of cavernous sinus and the obital fissure.	Motor nerve controlling the superior, internal, and inferior recti and the inferior oblique muscles of the eyeball. Also has parasympathetic innervation of the pupil sphincter and ciliary muscles.
4. Trochlear:	Trochlear nuclei on the floor of the cerebral aqueduct. Follows a path similar to oculomotor.	Motor nerve innervating the superior oblique muscle of the eyeball. Controls the outward and downward rotation of the eyes.
5. Trigeminal:		
Sensory Division:	Floor of fourth ventricle. Emerges over lateral pons to form three branches. (a) ophthalmic branch enters the superior orbital fissure; (b) maxillary branch passes through orbital tissue to infraorbital foramen; and (c) mandibular branch unites with motor division and leaves skull through the foramen ovale.	Ophthalmic branch carries sensations from cornea, ciliary body, iris, conjunctiva, nasal mucous membrane, eyelid, eyebrow, forehead, and nose. Maxillary is sensory for cheek, lower eyelid, nose and upper jaw, teeth, mucous of mouth. Mandibular is sensory for front of chin and temples.

TABLE 1—*Continued*

Cranial Nerve	*Origin and Course*	*Termination and Function*
Motor Division:	Follows mandibular branch of sensory division.	Anterior motor division controls mastication and lower mouth and skin. Posterior motor controls skin and muscles of posterior face including ear, mandibular joint, lower jaws, mouth, gums, and anterior 2/3 of tongue.
6. Abducens:	Floor of fourth ventricle. Leaves brain at lower pons and passes through lateral cavernous sinus to enter eye via superior orbital fissure.	Supplies the lateral rectus muscles of the eyeball and controls outward rotation of the eyes.
7. Facial:		
Sensory Division:	Geniculate ganglion cells of facial canal. Projects into the brain through the acoustic meatus between the inferior peduncle and olive.	Nerve for taste in the anterior 2/3 of the tongue and for the sensations of the soft palate. Also innervates salivary gland.
Motor Division:	Leaves brain at lower pons. Follows path similar to the sensory division and exits skull at stylomastoid foramen.	Motor supply to lateral muscles of face and scalp.
8. Acoustic:		
Cochlear Division:	Spinal ganglion of cochlea; peripheral fibers go to the organ of Corti; central fibers project into the inferior peduncle via the internal auditory meatus.	Terminates in the ventral and dorsal cochlear nucleus but a system of fibers ultimately projects to the superior temporal gyrus via the medial geniculate body of the thalamus. Hearing.
Vestibular Division:	Bipolar cells of the vestibular ganglion. Superior, inferior, and posterior branches end in the utricle, saccule, and ampule of the posterior semicircular canals, respectively.	Terminates in the medial, lateral superior, and spinal vestibular nuclei on the floor of fourth ventricle and in cerebellum. Sense of equilibrium of head and body.

TABLE 1—*Continued*

Cranial Nerve	*Origin and Course*	*Termination and Function*
9. Glosso-pharyngeal:	Passes from medulla to exit between the internal carotid artery and internal jugular vein.	Sensory for taste in posterior third of tongue and for the tonsils, pharynx, and soft palate. Motor for pharynx and stylopharyngeus muscles of throat.
10. Vagus:	Leaves brain between inferior peduncle and olive and passes through the jugular foramen to join cranial portions of #11. Continues via neck and thorax to the abdomen.	Sensory to posterior external auditory meatus, pharynx, larynx, thoracic and abdominal viscera. Motor to some regions of throat and to autonomic ganglia in thorax and gut.
11. Spinal Accessories:	Cranial portion rootlets leave brain at the side of the medulla and run below vagus to be joined by spinal portion which arise from motor cells in the anterior grey column of cervical segments of the spinal cord.	Cranial portion is motor to pharynx, larynx, uvula and palate. Spinal portion is motor to sternomastoid and trapezius muscles of posterior neck.
12. Hypoglossal:	Rootlets emerge in medulla, course through the hypoglossal canal of the occipital bone to form a nerve trunk, where they turn downward, then forward to the anterior neck and tongue.	Motor to the muscles of the anterior neck and tongue.

Since, in general, the brain develops along a caudal rostral axis, the development of the cranial nerves proceeds in an inverse numerical order. The topographical pattern of reflexive development mediated by the cranial nerves is correlated with the development of the cranial nerves.

The visual system, unlike other sensory systems, develops as an extension from the forebrain into the periphery. The optic nerve and the retina grow from the basal forebrain and differentiate within the eye by the eighth month of gestation. In this and many other respects, the visual system represents a special sense. It is highly organized in the periphery, and the eyeball represents a very specialized system of organization for carrying light to the photosensitive receptor cells, the rods and cones.

In addition, the eyeball contains a chain of neural elements that carry the neural impulse generated by the receptors to the visual cortex. There are three synaptic links in this anatomical chain. The receptor cells synapse with the bipolar cells which in turn synapse with the ganglion cells that make up the optic nerve. In the lateral geniculate body of the thalamus there is a third synapse when the ganglion cell processes meet the cells of the lateral geniculate body that make up the optic radiations to the visual cortex, the visual cortex being located in the most posterior portion of the cerebral cortex, the occipital lobe.

The microscopic development of the visual system has been studied very extensively (Eichorn, 1963). Within the retina, the cells begin to form layers about the seventh month of gestation, but the layered appearance of cells is not complete until about sixteen weeks after birth. The increase in layers is accompanied by deposition of myelin both from the thalamus to the peripheral eye and to the cortex. This developmental pattern is far from complete by birth. Increases in the amount of myelin probably continue at a relatively high rate until some years after birth. The changes are reflected in increased visual acuity and possibly by increases in visual sensitivity.

Integrating Systems of the Brain. Reflexive reactions of a relatively simple nature are organized in the spinal cord and within the brain via the cranial nerves. However, the elaboration of more complex behaviors necessitates a higher order of interaction and integration than has heretofore been discussed. Integrating neural systems must exist in order to perform the diversity of behaviors evidenced in the human organism. The evolution of the brain has resulted in several such systems, but the following discussion will be limited to only three of them. They are similar in that all three represent a high order of sensory motor integration, but they are markedly different in terms of their respective levels of integration.

Reticular System. The reticular formation of the brain stem constitutes one of the phylogenetically oldest of the integrating systems. As an anatomical structure, the reticular system is located in the central core of the brain (see Figure 2). Its cells are highly intermixed into a reticular or netlike arrangement. Such a network of cells gives the system many possibilities for gross reflexive acts, but it does not easily allow particular refinements of motor acts. Thus, in simple organisms, or in the foetus, reflexive actions first show themselves as massive responses, e.g., gross swimming movements or responses which orient the organism away from the source of painful stimuli.

In the adult, the reticular system of the brain stem has three primary functions. First, it regulates the degree of gross motor tension observed in the musculature of the organism via facilitation or inhibition of activity in the motor pathways which lie ventral to the reticular core. Second, the reticular formation regulates sensory integration by similarly altering activity in the collaterals from the primary sensory pathways which project into the brain. Third, the reticular core functions in the regulation of cycles of sleep and wakefulness. With regard to the last of these capacities, Moruzzi and Magoun (1949) have related activity in the reticular core to the electrical activity of the brain. Animals with extensive damage to the reticular system are somnolescent and can only be awakened with intense sensory stimulation. Electrical stimulation of the reticular core, on the other hand, will alert animals quite readily, even when the level of stimulation is lower than that applied to other parts of the brain in order to produce an alert state.

Structurally, the core of the reticular system is sufficiently well developed at birth to cope with the first two of its above mentioned functions, but the more remote parts of this system, which seem to be related to the levels of vigilance and sleep, are still not completely functional. These characteristics must await further development in the forebrain.

The Rhinencephalon and the Limbic System. All of the various integrating capacities of the reticular core are limited. As evolutionary pressures demanded more complex integrating mechanisms, the brain evolved more versatile levels of functional organization. In the lower order vertebrates that live in the water or close to the ground, the sense of smell, as a rudimentary distal detecting system, gave the organism a decided advantage in searching out food or finding a mate. But a distal detector also required a higher level of coordination between the input signal and the output acts. This was particularly true for animals that moved out of the water and gained a greater degree of locomotor versatility. The development of the forebrain, particularly the rhinencephalon (Gr: rhis, nose), is due in large part to the evolutionary escalation of versatility as a requirement for survival. The olfactory system thus forms the basis of the rhinencephalon and the related limbic system. The current speculation is that these areas are no longer primarily olfactory in function in man, but have subsumed other functions more related to the expression of emotional behaviors.

From the olfactory bulb, nerve fibers project to the higher centers of integration in the rhinencephalon. In general, these structures represent the earliest appearance of cortical tissue in the nervous system. Structurally, the rhinencephalon and the related limbic region form a ring around the center of the forebrain (see Figure 3). Nerve fibers from the olfactory bulb project to the prepyriform cortex and the corticomedial division of the amygdaloid complex, structures located on the interior surface of the temporal lobe (see Figure 3). In turn, these areas have fiber tracts which connect with secondary olfactory areas: the basolateral division of the amygdala and the frontotemporal cortex. There is also a third projection area, which has connections with the secondary but not with the primary projections from the olfactory bulb. The areas in the brain which constitute this third system are the entrorhinal, retrosplenial and cingulate cortex, and the Ammons formation, composed of the hippocampus and adjacent structures.

There is an anatomical distinction that is generally made between the structures which constitute the rhinencephalon and those which make up the limbic lobe. Those areas which receive direct projections from the olfactory bulb are generally considered to be parts of the rhinencephalon. Those secondary and tertiary regions are regarded as parts of the limbic lobe or limbic system.

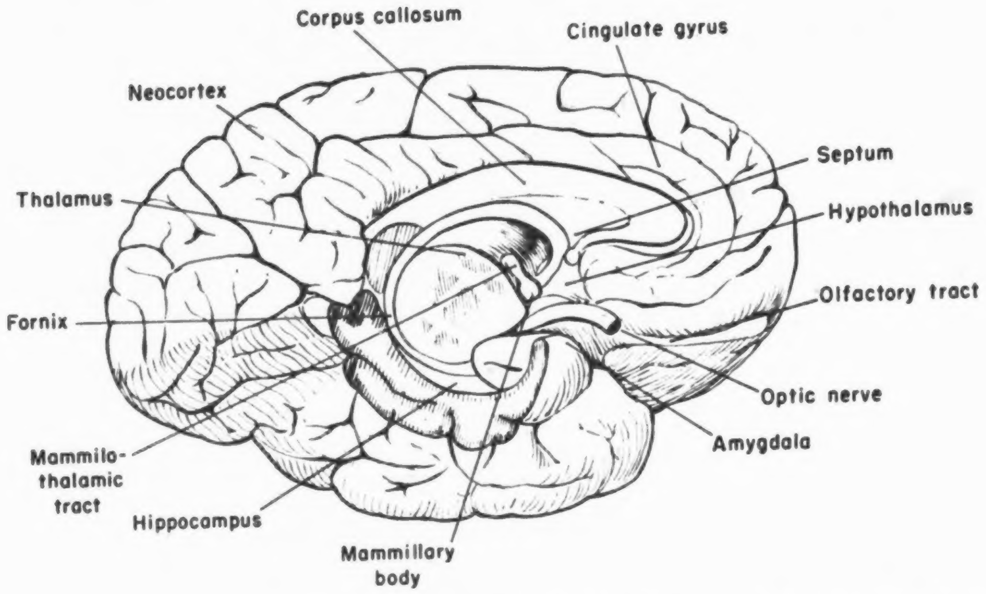
Corpus callosum
Cingulate gyrus
Neocortex
Septum
Thalamus
Hypothalamus
Olfactory tract
Fornix
Optic nerve
Amygdala
Mammilo-thalamic tract
Hippocampus
Mammillary body

FIGURE 3. Structures of the rhinencephalon in the adult brain. The brain stem, cerebellum, and portions of the medial surface of the temporal lobe have been removed in the drawing to show the origin and path of the fornix (Ranson and Clark, 1959).

Many of the structures of the rhinencephalon and limbic lobe have direct connections with the hypothalamus, a diencephalic structure which sits at the base of the brain and is extremely important in the regulation of many functions necessary for the survival of the organism. Because of the high degree of interaction between these areas and the hypothalamus, we will briefly discuss some of the hypothalamic functions, then return to a more detailed presentation of the functional attributes of the limbic system.

In a very broad sense, the hypothalamus represents an integrating center in the visceral system. Its importance in behavior is apparent when one considers that the hypothalamus coordinates and regulates the activities of the autonomic nervous system. Indeed, recent work indicates that this area of the brain may constitute the organizing centers for pleasure and pain (Olds, 1955) and the origin of many emotional reactions (Hess and Akert, 1955).

Embryologically, the hypothalamus develops in man on the interior fold of the telencephalon (see Figure 1) and extends from the region anterior to the tegmentum of the brain stem to the optic chiasma on the floor of the third ventricle (see Figure 2). It is a highly nucleated structure. In the rat, the nuclei of the hypothalamus continue to develop until shortly before birth (Coggeshall, 1964). The same is probably true for man. Myelination does not appear in this area until about ten days of postnatal growth in the rat. Thus it is probably functional at birth as a homeostatic reflexive mechanism, but its interactions with other centers and its efficiency of operation do not occur until later in life.

The functions of the hypothalamus are many and varied. Minute destruction of some of its parts produces a great variety of abnormal syndromes, as evidenced by experimental studies on animals. The most anterior area appears to control the regulation of body temperature. Other hypothalamic nuclei regulate some of the endocrine activities of the pituitary gland (or hypophysis). Still other hypothalamic nuclei seem to be involved in the refinement of the peripheral and visceral reactions associated with strong emotional states. Damage to these areas produce tachycardia, pupil dilation, piloerection, elevated blood pressure, and, in experimental animals, a full rage reaction directed at sources of minor irritation. Rather slight damage to other areas results in abnormalities in the regulation of food intake, and excessive overeating (hyperphagia) and inactivity become the predominant behaviors. This syndrome often includes components of rage. Even other areas, when damaged, produce a cessation of food ingestion (aphagia).

The involvement of the hypothalamus in the elaboration of limbic system activity is evidenced by the afferent paths that project to various areas of the hypothalamus. These anatomical interactions give the hypothalamus a potential functional interaction with other integrating systems in the brain, particularly the reticular formation and the limbic system. Developmentally, the hypothalamus is probably functional before the limbic system. Myelination of the hypothalamus precedes myelination of the fiber tracts from the fornix or the median forebrain bundle, two important sources of input to the hypothalamus. Kling and Coustan (1964) chronically implanted electrodes in both the hypothalamus and the amygdala of cats at birth. Electrical stimulation of these areas resulted in numerous indications of visceral activity, such as pupil dilation, piloerection, hissing, etc. Usually these reactions to stimulation could be elicited by hypothalamic stimulation earlier than by amygdaloid stimulation. This would suggest primacy of the hypothalamus over rhinencephalic areas in the control of visceral activity.

Virtually all of the structures of the limbic system have been implicated in some way in the regulation of behavior, particularly behavior best described as "emotional." Stimulation of the amygdala results in rage or docility reactions in wild animals (McLean and Delgado, 1953; Anand and Dua, 1955). Destruction of the amygdala will produce a docile animal if the animal is wild and aggressive prior to the operation (Goddard, 1964). Amygdalectomies in human patients have been reported to result in a "marked reduction in the emotional excitability and normalization of the patients' social behavior and adaptation" in 85 percent of those who underwent the operation (Narabayashi, Nagao, Saito, Yoshida, and Nagahata, 1963).

The inferior horn of the hippocampus is also located in the temporal lobe. In subhuman organisms, damage to this area results in a distinct tendency toward preservation or repetition of the animal's ongoing activity (Kimble, 1963; Gol, Kellaway, Shapiro, and Hurst, 1963). This increase in activity may be due to an increase in the animal's reactivity to external environmental stimuli (Kamback, 1967). In humans, there is quite a different alteration associated with temporal lobe damage involving the hippocampus. Milner and Penfield (1955) observed that patients with bilateral lesions in the hippocampal gyri showed a distinct memory loss for material they had learned only moments before. Furthermore, stimulation of the hippocampus after removal of the temporal cortex which surrounds the hippocampus evoked memory of things the patient had experienced at some time in the past.

The destruction of other areas of the limbic system do produce marked alterations in emotional behaviors. The septal region, for example, seems to be intimately involved in the expression of rage. Brady and Nauta (1953) have suggested that the septal area exerts an inhibitory effect on rage responses elicited by hypothalamic structures, since lesions in the septum produced a lower threshold for rage. Septal stimulation, on the other hand, produces pleasurable effects, since stimulation can serve as a reward for the behavior of man and other animals (Olds, 1955; Bishop, Elder, and Heath, 1964).

Although it is not yet possible to specify in detail the nature of the interaction between the hypothalamus and the limbic lobe, it is apparent that these structures play a significant role in emotional behavior. The existence of the multiplicity of fiber tracts interconnecting these structures gives the brain a multiplicity of potentially available routes for integrating the behavior sequences observed in emotional behavior. This does not mean that these structures are not involved in the regulation of other behavior processes, but it would appear that their primary function is to regulate emotional reactions to environmental events.

Integration in the Cortex. A third and possibly the highest order of sensory motor integration exists in the neocortex. The cerebral mantle to the brain is the primary neural structure that discriminates mammals from nonmammals, and its importance for the organization of sensory information and the elaboration of motor behavior is paramount to a complete understanding of the brain. This structure gives man and other mammals their unique capacity for complex behavior.

The neocortex developed as a neural apparatus which allowed finer degrees of sensory integration and motor control. The need arose as evolutionary pressures made organisms more and more dependent on the use of their distal receptor organs and fine control of their musculature.

Like the eye, the other sensory systems of the body, with the exception of olfaction, also project to the posterior portions of the cerebral cortex. Auditory fibers project from the medial geniculate body of the thalamus to the supratemporal plane of the temporal lobe. Somesthetic sense fibers project from the ventrolateral nuclei of the thalamus to the postcentral gyrus of the cortex. Figure 4 shows a schematic representation of the location of sensory and motor areas in the neocortex.

As in the visual system, the other sensory systems project from the periphery to the cortex and maintain a topical representation of the

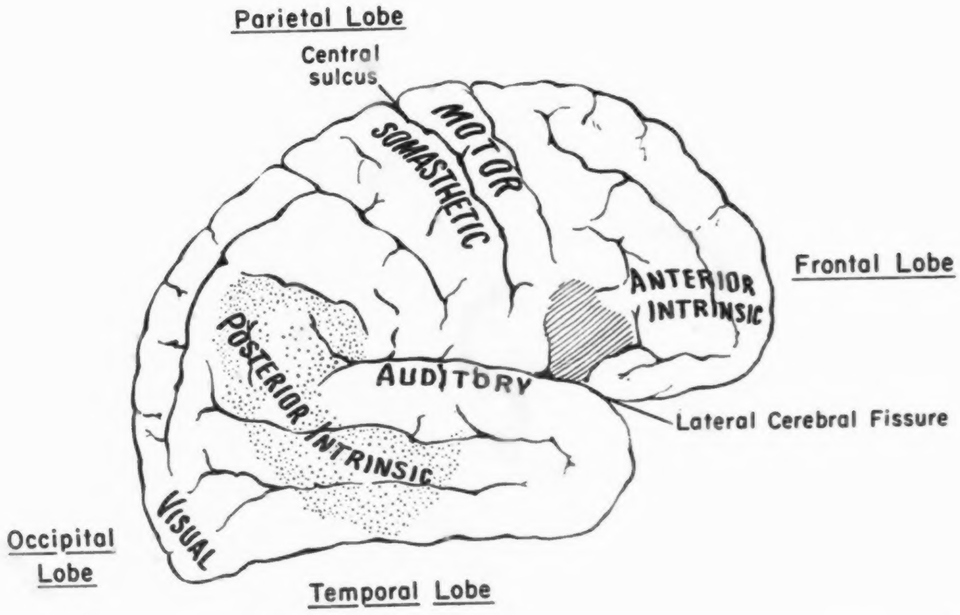
Parietal Lobe
Central sulcus
MOTOR
SOMASTHETIC
ANTERIOR INTRINSIC
Frontal Lobe
POSTERIOR INTRINSIC
AUDITORY
Lateral Cerebral Fissure
VISUAL
Occipital Lobe
Temporal Lobe

FIGURE 4. A lateral view of the adult cortex. The shaded region in the anterior part of the brain represents Broca's area. The stipled region in the posterior brain is Wernicke's area (Penfield and Roberts, 1959; Pribram, 1960).

peripheral sites of origin of the sensory nerves. That is, nerves from a particular place on the periphery project to a particular place in the cortex. This has been demonstrated for both the sensory systems and the pyramidal motor system of the precentral gyrus, which represents a return path from the cortex to the striate muscles of the periphery.

On the output side, there are at least three systems of motor control in the body whose fiber tracts arise from some area of the cortex anterior to the central gyrus. Their degree and level of control over motor actions differ appreciably. The first of these systems is the pyramidal system, so named because the nerve fibers in this system arise from giant pyramid shaped cells in the cortex. These fibers project downward to the spinal cord where most of them synapse on the opposite side of the spinal cord with the motor neurons that innervate the striate muscles of the periphery. This system also maintains a topical relationship between the brain and the periphery, and, like the somesthetic sensory system, a relatively large amount of cortical area is allotted to those portions of the periphery which require a great amount of fine motor control.

The second system, closely interrelated to the first both functionally and anatomically, is called the extrapyramidal system. As the name implies, it is composed of all the central components of motor control that the pyramidal system is not. These include the basal ganglia, subthalamic nuclei of the diencephalon, the reticular system of the brain stem, and the very important cerebellar cortex. Functionally, this system regulates the fine actions associated with coordinated movements, and lesions in this system can produce a variety of motor symptoms characterized by hyperkinetic syndromes, e.g., Parkinson's disease.

A third efferent system from the cortex which is part of the extrapyramidal system, but which deserves special attention for theoretical reasons, is the corticofugal system. This system has been defined primarily by electrophysiological procedures. Stimulation in the cortex will produce a wide variety of electrical changes in the reticular core of the brain stem, and will alter the electrical responses elicited by peripheral stimulation that reach the cortex. At present, it is not known how such a system functions, but it is believed that it exerts control over the peripheral sensory motor systems, and that through its interaction in the reticular system it can modify the kinds of stimuli toward which the organism will respond. Such a mechanism would be an important one in the regulation of the attentional states of the organism.

Although the sensory and motor regions occupy a large part of the cerebral cortex, there are portions of the cortex about which relatively little is understood. In part, these areas represent secondary or even tertiary areas of sensory or motor function, and they are generally in close proximity to the primary sensory areas.

The term intrinsic has been applied to other nonspecific projection areas of the cortex, because their connections to and from the thalamus involve the internal core of this structure (Rose and Woolsey, 1949). Pribram (1960) has suggested that these areas, the so called association areas, can be divided into two intrinsic cortical regions. The anterior intrinsic system is the frontal portion of the neocortex and receives its

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projections from the medial nuclei of the thalamus. The posterior intrinsic system receives projections from the dorsal thalamic group in the parietal lobe and from the posterior, lateral, and dorsal lateral thalamic nuclei in the inferior temporal lobe. There is a great amount of experimental data on the functions of the frontal lobe, and much of it indicates that this cortical region is involved in the ability to maintain attentional states and to direct planned sequences of behavior. The posterior intrinsic areas may mediate the ability to program and store information in generalized categories (Pribram, 1960).

Though the neocortex is the most conspicuous and most extensive part of man's brain, its function remains a mystery. As indicated above, the cortex is a final projection point of most of the sensory systems and is the starting point of systems of voluntary motor control. These facts alone are enough to imply that its function is to integrate input and output systems for the organism. In addition, its cellular structure implies that it is a region of the brain with capabilities for maximizing potential interrelationships between neural elements, for there are an estimated ten billion cells in the neocortex alone.

Cortical Role in Speech. Perhaps some insight into cortical function can be gained by briefly examining the role of the cortex in the production and perception of speech. Work in this area, accumulated from a great number of clinical cases, is reviewed in detail by Penfield and Roberts (1959).

Aphasia (Gr: a—, not; phasis, speech) is a clinical syndrome characterized by either a loss of the power of expression by speech or writing, or a loss in the ability to comprehend spoken or written language. The former deficit is a special case of apraxia (Gr: prassien, to do), a loss of the ability to perform purposeful movements, and is generally referred to as ataxia (Gr: ataxia, disordered), or expressive aphasia. The latter aphasic syndrome is a special case of agnosia (Gr: gnosis, a recognizing), a loss of the capacity to recognize the importance or meaning of sensory stimuli. Both apraxia and agnosia can be applied to a much wider range of clinical deficits than those involving speech. For example, the inability of a patient to extend his hand on command would be apraxia, while the failure to recognize a familiar visual object would be visual agnosia.

The production of speech is a complex motor act involving the temporal integration of a variety of movements of the muscles of the larynx, lips, tongue, mouth, etc. If the cortex is damaged in the region of the frontal lobe superior to the sylvian fissure and slightly anterior and ventral to the primary motor cortex, the individual suffers a loss in his ability to form words and to speak, though there is not an associated loss in the capacity to move the tongue, lips, etc. Stimulation of this area also disrupts the ability to speak. This region of the brain, called Broca's area, is located in the secondary motor cortex, which controls the movements of throat, tongue, jaw, and lips. This would suggest that Broca's area integrates the sequencing of motor events that are necessary for the production of speech. The magnitude of the speech deficit is greatest if the brain is damaged on the side contralateral to the preferred side of the body, though if this area is damaged unilaterally in a child, there is probably a transfer of function to the opposite hemisphere.

A second major area that is functional in both the elaboration and perception of speech is located on the posterior part of the superior temporal lobe in the general region of the inferior parietal lobe and anterior occipital lobe. This region extends into the temporal lobe on the inferior fold of the superior temporal gyrus. It is called Wernicke's area, and damage to this region will result in loss in the individual's ability to understand speech as well as loss in the production of ideational speech, without associated deficits in hearing and locomotor activity of the speech apparatus. This area is located immediately ventral and posterior to the primary auditory receiving areas of the cortex.

The perception of speech requires a rapid sequencing of sensory events (sounds) that must be integrated over time. It is probably true that Wernicke's area performs this integrative function. The involvement of this area in the motor elaboration of ideational speech may be the result of the fact that the elaboration of speech patterns necessitates the formation of the ideas of sounds as they have been perceived as the first step in speaking. If this initial process is destroyed, the formation of words is necessarily disrupted. Broca's area and Wernicke's area are parts of the anterior and posterior intrinsic areas of the brain, as outlined by Pribram (1960). A more extensive discussion of language disorders can be found in the chapter by Horton in this volume.

The perception of speech represents a special case of the more general integration of motor acts and sensory stimuli that are accomplished by the neocortex. Thus it would appear that this part of the brain, the neocortex, gives mammals, particularly man, a capacity to form extremely refined motor acts and to resolve minute sensory discriminations, and consequently adds a considerable amount of flexibility to their behaviors.

Plasticity in the Developing Brain

The effects of damage to specific areas of the brain during development have only recently begun to be investigated systematically, and this work has been done almost exclusively in the postnatal organism. Though there is extensive clinical literature which indicates that brain damage inflicted during the early days of postuterine life will produce functional deficits that last a lifetime, much of this is ad hoc conjecture without a firm experimental basis. Indeed, studies which have experimentally produced lesions in the brain of the newborn suggest that the developing brain has a considerable amount of functional plasticity.

Scharlock, Tucker, and Strominger (1963) investigated the effects of bilateral ablations of the auditory cortex of infant cats on their ability to learn to avoid a shock by responding to tones which differed in duration. Though in the adult cat a similar lesion produced a marked deficit in this capacity, Scharlock, et al., found that the animals which received the lesion between seven and ten days of age showed no loss in their ability to learn the problem when tested as adults, i.e., they did not differ from unoperated controls.

Doty (1961) has reported that following the ablation of the striate or extrastriate (visual) cortex in newborn cats, there is no loss in their ability to perform a visual pattern discrimination, though adult animals with similar lesions cannot perform this task. Tucker and Kling (1966) have found that cats with similar striate lesions inflicted prior to two months of age do not lose their ability to discriminate the temporal sequence of light flashes. Adult operated animals could not acquire this discrimination. These experiments would indicate that the sensory areas of the neocortex can undergo a considerable amount of damage early in life, without affecting the organism's ability to learn sensory discriminations.

But what of higher mental processes? Do the parts of the brain which mediate processes other than sensory processes show the same degree of plasticity? It is a well established fact that bilateral destruction to the granular cortex of the frontal lobe of the monkey will impair the efficiency of performance of a delayed response task. In this task, the monkey is presented with two visual stimuli, and the presence of the reward behind one of these stimuli is indicated. An opaque screen is then lowered between the monkey and the stimuli for some duration, and the monkey must then delay his response for a time determined by the experimenter. This task requires that the monkey not only learn to discriminate the correct stimulus but also remember which stimulus is associated with the reward. Tucker and Kling (1965) found that newborn monkeys with frontal lobe damage could learn to delay their response up to forty seconds (the maximum delay period tested). The adult operated animals could not perform the task if the delay was beyond five seconds. Thus it would appear that, like sensory functions mediated by cortical structures, "higher" intellectual functions can be reorganized during development.

Apparently similar effects can be observed following damage to subcortical structures. Kling and Green (personal communication, 1966) compared the effects of fear responses to threatening stimuli in infant and adult monkeys subjected to bilateral amygdalectomies. The adult animals showed the typical temporal lobe syndrome, i.e., reduced fearfulness, but the animals which received amygdalectomies in infancy were as fearful as their unoperated controls. Whether this relationship would hold for other subcortical structures is not known.

Though this work rather clearly indicates that damage to specific structures in the brain in infancy does not produce the same extensive losses as similar lesions inflicted on the adult, there are numerous questions which remain unanswered. Are these effects age specific? Are these effects the same across all species? What is the nature of the reorganization process within the brain? Might not the effects of the lesion in the infant show up under other stimulus situations than those employed? The answers to these and similar questions await further research.

Role of Environment in Development of the Nervous System. Haywood and Tapp (1966) have reviewed an extensive body of psychological literature which indicates that the environment within which a developing organism is raised can produce general alterations in behavioral predispositions in adulthood. Furthermore, the nature of the behavioral change is dependent on the age at which the organism encounters particular environments. Though this is an old axiom in psychological theory, it has only recently been subjected to experimental investigation, and there is some indication that effects are mediated by demonstrable neural or hormonal changes that accompany the environmental experience. The question raised by the Haywood and Tapp review, of relevance to this volume, is, "What is the nature of the interaction between the environment and the effects of brain damage in the developing organism?" Unfortunately, this question has not been thoroughly investigated.

In the adult organism there appears to be a good deal of interaction between recovery of function and the environment in which the operated animal is maintained between the operation and the test of functional capacity. Meyer, Isaac, and Maher (1958) trained a group of rats on a light avoidance response. Following bilateral removal of the visual cortex, the rats lost the habit. If, however, the occipital cortex was removed in a two stage operation, there was a high degree of interaction between the recovery of function and the environment. If, during the interval that separated stage one from stage two, the animals were kept in darkness, they lost the visual habit. If they were kept in their home cage under normal living conditions between unilateral operations, the animals showed no loss of habit and were similar in performance to sham operated controls. Petrinovich and Bliss

(1966) have replicated the essential features of these results, but Thompson (1960) found a recovery of function only when animals were retrained on the task during the interval between the unilateral operations. Isaac (1964) has further demonstrated that the recovery of function is enhanced by extending the time interval between operations, or by subjecting the animals to greater amounts of sensory stimulation. These experiments indicate that a recovery of function can be greatly enhanced by environmental stimulation. However, such an effect has not been demonstrated with bilateral removal of any part of the brain.

The nature of the animal's experience preceding the operation may also be an important contributing factor to the nature of the postoperative deficit. Smith (1959) compared the effects of cortical lesions in rats raised in either a complex, enriched environment or in an isolated, restricted environment. When the animals were tested in a maze on a series of perceptual learning problems following cortical lesions, the deficit produced by the lesions was somewhat greater in the animals raised in the complex environment. The magnitude of this effect was dependent on the location of the lesion within the cortex. A lesion in the posterior visual cortex produced a greater deficit in the rats raised in an enriched environment than it did in those reared in the home cage. Wittrig (1962) has found the opposite trend, i.e., rats raised in an enriched environment were less affected in learning a Lashley III maze by an occipital cortical lesion than those raised in an impoverished environment. Wittrig's result was not statistically significant and is only suggestive. In both experiments, the enrichment procedure considerably improved maze learning.

Kling and Green (personal communication, 1966) compared the effects of amygdalectomies on rats reared either in social isolation or with their mothers. The isolated animals showed fearfulness of a strange observer or a strange situation, as had been demonstrated by Harlow (1958), but this fearful behavior was not diminished by amygdalectomy, though the operation did reduce fearful behavior in normally reared monkeys. In the experiments of both Kling and Green (personal communication, 1966) and of Smith (1959), the effect of the rearing condition produced a much stronger effect than the lesion.

Only one published experiment has investigated the effect of environmental enrichment on the recovery of brain damage inflicted on the young. Swartz (1964) made lesions in the neocortex of newborn rats, then raised them in either an enriched or a normal laboratory environment until they were 94 to 96 days of age (early adulthood). All animals were then maintained under normal laboratory conditions until they were tested on a series of problem solving tasks in a maze. The animals raised in the rich environment made fewer errors than those raised under normal laboratory conditions. The rats with lesions also made more errors than the controls. Most important, though, was the observation that environmental enrichment procedures reduced the magnitude of the deficit produced by the lesion by approximately 50 percent.

Though more research is necessary to investigate the interaction between experience and brain damage, the implication of all these experiments is clear. The environment can play a significant role in determining (a) the nature and extent of the deficit produced by brain damage, and (b) the nature of the recovery following damage to the developing brain.

Summary and Conclusions

The material reviewed in this chapter provides the reader with an overview of the gross organization of the brain within the context of its development. It is apparent that the brain develops in stages that are delineated by the appearance of particular morphological characteristics. The neuroblasts of the embryo divide and migrate to their position in the brain, then originate those morphological characteristics which give the nerve cells their unique function in the regulation of behavior. These stages are not discrete, but overlap one another and appear at different ages in different parts of the brain. Damage to the brain during these periods generally represents damage to those cells which are developing most rapidly. This would suggest that similar causes of abnormal cell development might produce markedly different effects, depending upon the age of the organism at the time of brain insult. The functional manifestations of this damage might even be markedly different from one individual to the next.

The growth of the central nervous system is accompanied by the elaboration of functional capacities, evidenced by the behavior of the organism. The development of simple reflexive acts during the prenatal period increases the newborn's potential for survival, and the postnatal development of neural integrating systems allows the basic reflexive patterns to become modified by the experience of the organism. Though structural functional relationships have been investigated only in the development of some simple reflexes such as grasping, it is quite probable that similar patterns of development exist in higher order reflexes.

Research on three of the major integrating systems of the brain has made great strides in determining their function. The reticular system of the brain stem seems to regulate the general state of excitability of the nervous system. Rhinencephalic and related limbic structures provide the mechanisms for emotional tone through their interaction with the hypothalamus. The cerebral cortex gives the organism a greater capacity for spacial and temporal integration of sensory and motor events. As future work delineates the nature of the interactions between these systems and broadens our understanding of structural functional interrelationships, problems of diagnosis will be considerably reduced.

Damage to the developing brain constitutes a major problem for future investigation. Though it would appear that the brain of the growing organism manifests a considerable amount of plasticity during its development, there are numerous questions that need to be examined in detail before the idea of plasticity becomes an established fact. However, it is already clear that the interaction between early brain damage and the organism's experience is an important determinant of the nature of functional deficit.

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The Epilepsies

ANGELA T. FOLSOM

Epilepsy is a disorder—more accurately a group of disorders—which afflicts four to seven per 1,000 individuals. In the United States, then, there are 800,000 to 1,000,000 persons who have epilepsy. There has never been any census of epilepsy. It is not a reportable disorder. Where it has been reportable, epileptics have been driven underground because of shame and real or supposed restrictions. The best figures available in this country are those from the World War II draft. At that time, epilepsy or a history thereof was grounds for rejection. Five to six per 1,000 were rejected by draft boards or induction stations because of it. Granted, some may have been rejected on a faked history or mistaken diagnosis. We know, however, that many were inducted who either denied they had ever had epileptic episodes or were not aware that the episodes they had were epileptic. At the National Veterans Epilepsy Center in Boston, it was not infrequent to discover a history of epilepsy prior to service in the armed forces. Epileptics are now not automatically rejected but placed on limited service status. Epilepsy is a physiological phenomenon and has little to do with psychology insofar as its basic etiology is concerned. An underlying cerebral disturbance or dysfunction is a *sine qua non* of any epileptic seizure. A seizure —or epilepsy, which usually implies recurrent seizures—is the observable symptom of an underlying electrical discharge in the brain. An abnormal discharge can take place without resulting in a seizure but a seizure probably does not occur without an abnormal discharge. There are, however, many psychological concomitants of epilepsy. Epileptic phenomena can be affected—either facilitated or suppressed—by psychological factors. The sociopsychological concomitants of epilepsy can be devastating, not so much because of what it is but because of what we think it is. By we, I mean the patient, his family, coworkers, employers, teachers, and friends. Further, there are some episodes which are a real problem in differential diagnosis. There are such things as hysterical seizures. There are patients who have both hysteria and epilepsy. There is a type of epileptic seizure, the psychomotor seizure, which is sometimes very difficult even for the epileptologist to differentiate from a dissociative fugue state.

The name epilepsy was first used by Avicenna in the eleventh century. It is the one of many terms for this disorder which has survived. The Greek word from which it is derived means a condition of being overcome, seized, or attacked, a condition keenly felt by patients and observers. It is a frightening experience to observe a person suddenly change from his usual self to something quite different, sometimes almost animalistic. Beliefs as to what the person is seized by have ranged from God to the devil. Common or popular names for seizures in many languages denote emotions of fear, disgust, and the eerie. These beliefs and folklore have left their mark on our ideas about the epileptic in the psychological and psychiatric literature, in our laws, and in the seizure patient's reaction to his illness.

I want first to present some of the more common types of seizure patterns and some electroencephalographic correlates of brain dysfunction, along with some consideration of etiology. I shall be mainly concerned with sociopsychological considerations in the second section.

Classifications and diagnostic labels vary from place to place. Table 1 presents two classifications by two major workers in epilepsy: Lennox

TABLE 1

Different Ways of Classifying Epilepsy

I. *Lennox's Classification*
    A. Petit mal triad
        1. Petits (pure petit mal—pyknolepsy)
        2. Myoclonia (myoclonic or myoclonus)
        3. Atonic
    B. Convulsive triad
        1. Generalized (grand mal)
        2. Focal (localized, partial)
        3. Jacksonian (Rolandic)
    C. Temporal lobe triad
        1. Automatic
        2. Subjective
        3. Tonic focal
    D. Automatic (periventricular)
    E. Unclassified or combined

TABLE 1—*Continued*

- II. *Penfield's Classification*
  - A. Focal cerebral seizures—focal epilepsy—symptomatic seizures
    - 1. Somatic motor
    - 2. Sensory
    - 3. Automatic
    - 4. Psychical
    - 5. Automatism
  - B. Centrencephalic seizures
    - 1. Petit mal
    - 2. Myoclonic petit mal
    - 3. Grand mal
    - 4. Petit mal automatism
    - 5. Psychomotor automatism
  - C. Cerebral seizures
    - 1. Origin of discharge unidentifiable or etiology of seizures
    - 2. Extracerebral
- III. *Other classification often used in clinical practice*
  - A. Clinical seizure type qualified by etiology
    - 1. Etiology unknown: terms used
      - a. Idiopathic
      - b. Cryptogenic
      - c. Essential
      - d. Genetic
      - e. Metabolic
    - 2. Etiology within the brain: terms used
      - a. Symptomatic
      - b. Acquired
    - 3. Etiology outside the brain (disease or agent named)
      - a. Grand mal seizures, idiopathic
      - b. Psychomotor seizures, symptomatic, left temporal lobe tumor
      - c. Idiopathic epilepsy manifested by (type of seizure)
      - d. Chronic alcoholism associated with withdrawal seizures

in Boston, and Penfield in Montreal. A more recent classification can be found in *Epilepsy: a review of basic and clinical research (*Department of Health, Education, and Welfare, 1965). There are a few descriptive terms for seizure patterns which have a wide usage—grand mal, petit mal, and psychomotor. Generally, the fewer the terms used, the less the concern about epilepsy and brain function. In many primarily psychiatric hospitals, classification into major and minor seizures suffices, or grand mal may be used to refer to any convulsive seizure and petit mal for any nonconvulsive seizure.

I am laboring the point of meaningful diagnostic terms for two main reasons. One is a very practical reason: if you do not know the referents for a diagnostic label, you do not know what behaviors to expect. Secondly, this variation in terms produces difficulties in the evaluation of published findings unless the investigator makes explicit the referents for the terms he is using. The term epilepsy has little meaning any more as an entity other than to indicate a paroxysmal recurrence of symptoms of brain origin The term epilepsies is more appropriate in the consideration of etiology, brain function, psychological factors, and treatment. I shall try to make some of these referents more explicit.

Penfield is a neurosurgeon and the horse he rides in his classification is that which proves useful to him, localization within the brain. His major categories refer essentially to the origin of epileptic discharge: cortical in the first group, highest level of the brain stem in the second group, and of unknown origin in the third group. Under each he lists types of seizure phenomena which occur as a result of initial discharge in these areas. His etiological terms are two in number—symptomatic we can identify the cause of the discharge, and cryptogenic: we cannot (now) identify the cause.

Lennox was one of the most humane clinicians working in the field of epilepsy. His classification reflects his clinical investigations over many years (his publications extend from 1922 to 1960) into a broad spectrum of factors he hoped would lead to some answers for the origin and cure of epilepsy. His petit mal triad is based on brief duration of seizures, early age of onset, and certain electrical discharge similarities in the EEG. His convulsive triad is based on motor seizure behavior, and his temporal lobe triad, on findings of a temporal lobe EEG focus. His etiological terms reflect his beliefs in the cause of epilepsy over the years—metabolic, genetic, and essential—and are used today as essentially equivalent to cryptogenic and the older term, idiopathic. Lennox preferred the term acquired, and even organic to symptomatic, both of which get him into difficulties. They all generally refer to the identifiability of the cause of seizures, or, in other words, to the cause of the excessive neuronal discharge of which the seizure is a symptom.

Before specific seizure patterns are discussed, there are a few more terms and concepts which should be clarified. Seizures, attacks or fits are observable phenomena to the layman. To the neurologist they are a symptom of neuronal discharge. Some symptoms may be subjective or observable only to the patient. Aura is the term used for these subjective phenomena which precede observable behavioral phenomena. They may be vague or definite feelings or vivid visual, auditory, or olfactory experiences, typically with a brief duration, which patients learn are usually followed by a blackout. Sometimes the subjective symptoms are the only manifestation of the seizure discharge. The term prodrome usually refers to mood change which in some patients may precede for days a seizure proper—moodiness, irritability, etc. A post epileptic or postictal (ictus is another term for seizure) clouded state, relatively rare in the entire epileptic population, may follow a seizure and implies anything from a severe confusional state to an outright psychotic episode which may last for weeks. Status is a term used for repeated seizures without regained consciousness between seizures.

There are three main seizure patterns: petit mal, grand mal, and psychomotor.

Petit Mal—a Disorder of Childhood

The primary characteristic of a pure petit mal seizure is a lapse of consciousness coming on abruptly and usually lasting less than 15 seconds. It disappears as quickly as it appears. To the observer there is a sudden immobility, a vacant look to the eyes, and a slackness or freezing of a blank facial expression. This is sometimes described as a "mean" look (see Figure 1). If the child is holding anything, he may drop it. There is occasionally rhythmic eye blinking, and, in a small proportion of these children, rhythmic movements of the arms or legs, always bilaterally. These patients do not fall or lose urinary control.

The patient has complete amnesia for the episode itself. He may become aware that something has happened when he drops things

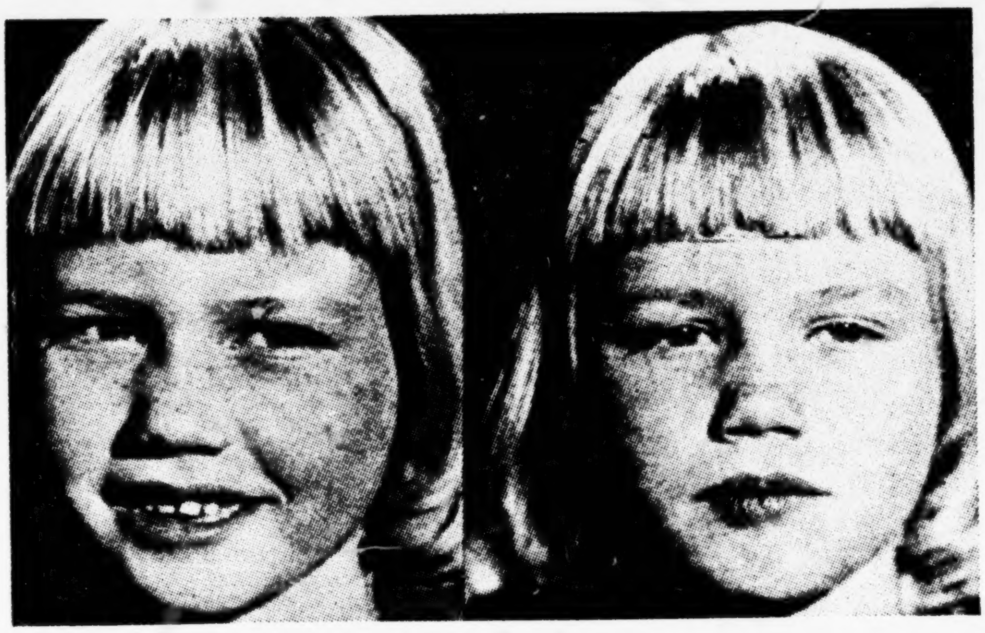

FIGURE 1. Child subject to petit mal attacks. Normal appearance on left; appearance during a petit mal attack on right.

frequently, when he misses parts of conversation, and most important for the child, by other persons' reactions to it.

Petit mal seizures usually start in the 4 to 12 year old age group and disappear usually by age 16 to 18. Rarely does an adult have pure petit mal seizures; when he does, he usually has other types of seizures also.

Parents often regard these episodes as tics, clumsiness, or purposeful inattention. When these seizures occur many times a day (and there may be up to a hundred in a day if not treated), the child is likely to present some kind of a problem in the classroom. Such children are often referred to clinics with data suggesting incorrigible day dreaming, inability to pay attention, or periodic unresponsiveness. This is not meant to imply a need for a fervent hunt for epilepsy among all students with these symptoms. I am suggesting, however, that certain earmarks of these symptoms may indicate petit mal seizures, particularly if these occur in a total picture of otherwise generally adaptive behavior. Individual talks with the child or observation during study periods may help in supplying pertinent material on the basis of which appropriate diagnostic procedures can be instituted.

From an electrophysiologic point of view, the pure petit mal seizure has fairly well established correlates, at least as evaluated by the electroencephalogram, or EEG. The EEG is a record of the electrical activity of the cerebral cortex via electrodes placed on the scalp. What it actually records is the difference in potential between two areas. This may be between an area of the brain and a common reference point, usually the ear in monopolar recordings, or between two areas of the

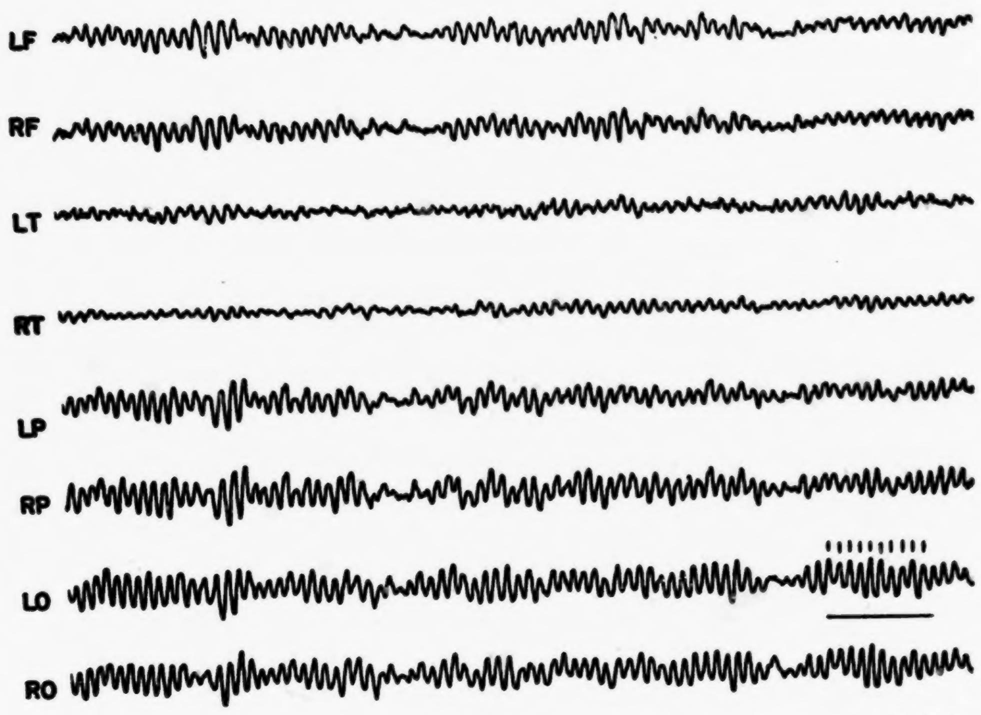

FIGURE 2. Example of a normal adult EEG recorded during resting wake, with 10 per second activity in all leads. Note higher amplitude in posterior leads, frequently the only leads from which alpha activity is recorded. Frequencies ranging from 8 to 12 per second are considered normal for any age. Letters on left indicate areas from which activity is recorded. L = left, R = right, F = frontal, T = temporal, P = parietal, O = occipital. Ear as common reference point.

brain in bipolar recordings. The more commonly used clinical EEG machines record electrical activity from eight sources simultaneously, and are known as eight channel machines. Sixteen channel machines at present are used more frequently in research work than in clinical work. The electrical impulses are greatly amplified and transmitted to the appropriate channels to activate pens on paper moving at a constant rate of speed, thus producing the electroencephalogram.

EEG's change with age, reflecting the development of the brain, and with level of consciousness. See Figure 2 for an example of a record obtained during resting wake and Figure 3 for one obtained during light sleep.

Figure 4, in contrast, is a recording of an 11 year old girl subject to petit mal attacks. Such an EEG contains regular three per second spike

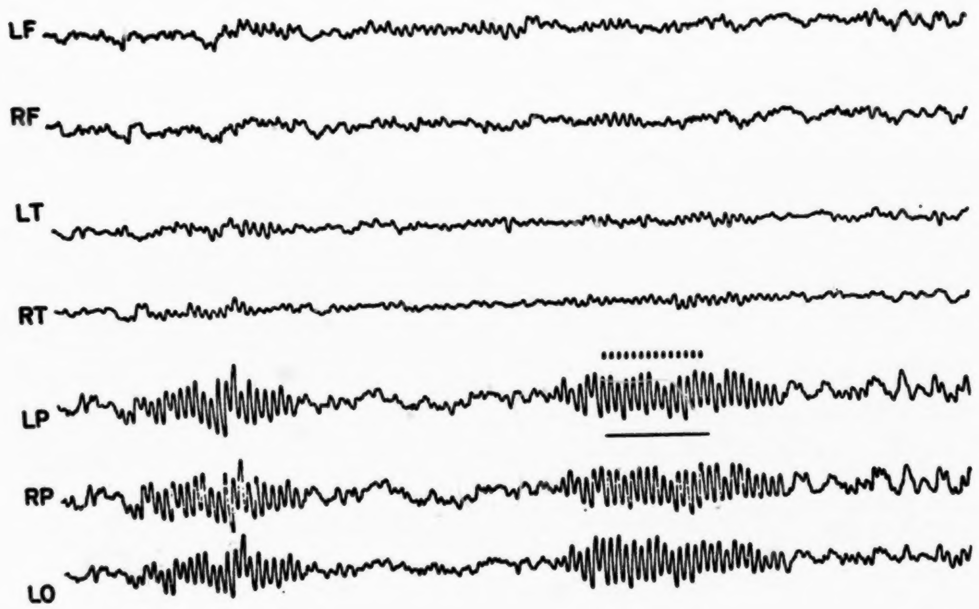

FIGURE 3. Example of a record during light sleep. Note the 14 per second spindle activity prominent in parietal and occipital areas. Letters and common reference point same as for Figure 2.

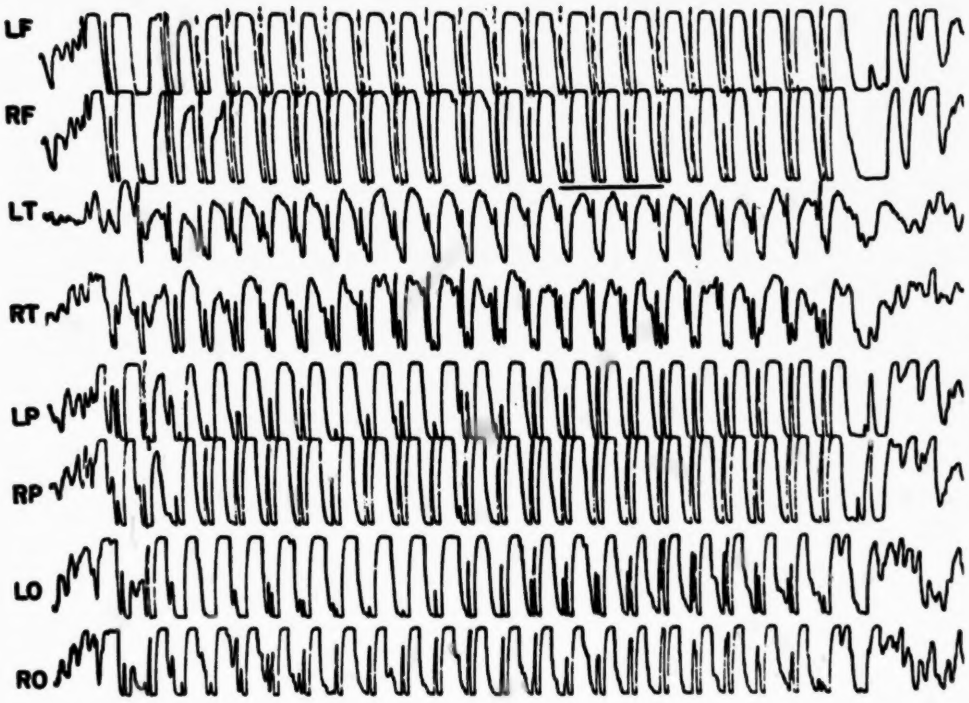

FIGURE 4. Example of 3 per second spike dome activity commonly seen in patients subject to petit mal seizures, with some multiple spikes in the temporal and occipital areas. This was the EEG of an 11 year old girl with a history of episodes of blinking eyes and/or staring. Letters and reference point same as in Figures 2 and 3.

and slow wave activity, referred to as spike dome or dart dome, bilaterally. The amplitude of the discharge is greatly increased over the normal background activity.

I have stated that this three per second spike dome EEG pattern is strongly associated with petit mal seizures. It occurs invariably during a seizure, and in about 80 percent of patients in the interseizure period. This contrasts with its occurrence in less than 10 percent of patients with other types of seizures. In a study of 1,000 nonepileptic subjects it occurred four times. Findings of high association of this type of discharge in patients with petit mal seizures have led many electroencephalographers to label this petit mal discharge. The naming of a neurophysiological phenomenon (in this case, the EEG discharge) by a descriptive clinical term (in this case, the type of seizure) is not considered good practice by many epileptologists. For one thing, it implies a one to one relationship which simply does not exist. For another, the unsophisticated recipient of such an EEG interpretation may infer that the electroencephalographer is making a diagnosis of petit mal seizures. No EEG finding by itself can yield a diagnosis. It may be a substantial diagnostic aid and may legitimately stimulate inquiry into diagnostic possibilities, but it should never be the sole basis of a diagnosis. Like any other laboratory finding, it must be put into perspective with the total clinical picture and history.

Petit mal attacks may be elaborated into what are generally called automatisms. During these the patient may continue to walk or carry out previously initiated activity, but has amnesia for this period. The bilateral motor movements may be more obvious, involving the trunk as well as the extremities. Dr. Lennox includes these manifestations under his petit mal triad; Dr. Penfield lists them separately. Sometimes the motor component, usually absent or very minimal in pure petit mal, becomes more prominent. In such cases bilateral jerks of the eyes, face, neck, legs, and body muscles, usually in that order, appear, and are the principal manifestation of the seizure. These may become so severe that the patient falls. Occasionally there may be no evidence of loss of consciousness. Penfield nevertheless keeps them in the centrencephalic group because of the spike wave formations found in the EEG. These formations are not so beautifully rhythmic as in pure petit mal.

Pure petit mal rarely occurs before four years of age. It is rarely associated with neurological signs or detectable brain damage. Other variations described above, especially if they occur early (i.e. in the first four years), are more stubborn to treat medically and may have a history of cerebral insult—prolonged fever, difficult birth with possible anoxic effects, etc. They are not generally associated with gross neurological defects. Intellectually, children with pure petit mal have been reported as average or superior in intelligence. I have wondered whether such findings may not be a result of superior parents bringing such children to treatment more readily. These seizures are generally influenced by psychological factors: they are more apt to occur when the patient is inactive, inattentive, drowsy. Many children have a flurry early in the morning before they are fully awake. Active interest and alertness tend to suppress these seizures.

Automatisms

I would like now to discuss automatisms. This is a term which covers a wide variety of behavior and is usually the most obvious aspect of the psychomotor seizure. At one end of the spectrum, it may merely involve the continuing of normal activity already started—walking, running, typing, painting, or driving—without gross deviation. The types of activity which are continued are usually well learned activities about which the person does not usually have to think in order to carry out effectively. The other end of the spectrum is seen in what appears to be well coordinated, from a motor point of view, but bizarre and "kookie activity." It may appear purposeful, but very inappropriate: looking in bureau drawers, wiping another person's nose with one's tie, wandering in a distracted manner. Occasionally the behavior is aggressive. I have only known one such patient: he almost invariably reached for the neck of the nearest person and squeezed. As in most of these automatisms, and fortunately for the people near him, this was of short duration. Many persons during automatisms do react against restraint with fighting and hitting out. Unfortunately, restraint is often applied, and I have wondered whether this is one reason that epileptics with automatisms as part of their seizure have earned the reputation of being aggressive and violent. This should not be confused with a post-ictal clouded state during which the patient may be psychotic for hours or days.

With this type of seizure is often found a temporal lobe EEG abnormality, usually spike activity, and thus they have come to be known as temporal lobe seizures. (See Figure 5 for distribution of recording of these spikes, and Figure 6 for an EEG record.) This does not necessarily mean that the discharge originates in the temporal lobe, but rather that the temporal lobe is involved electrically in the elaboration of the seizure, and a temporal lobe focus is recorded electrographically.

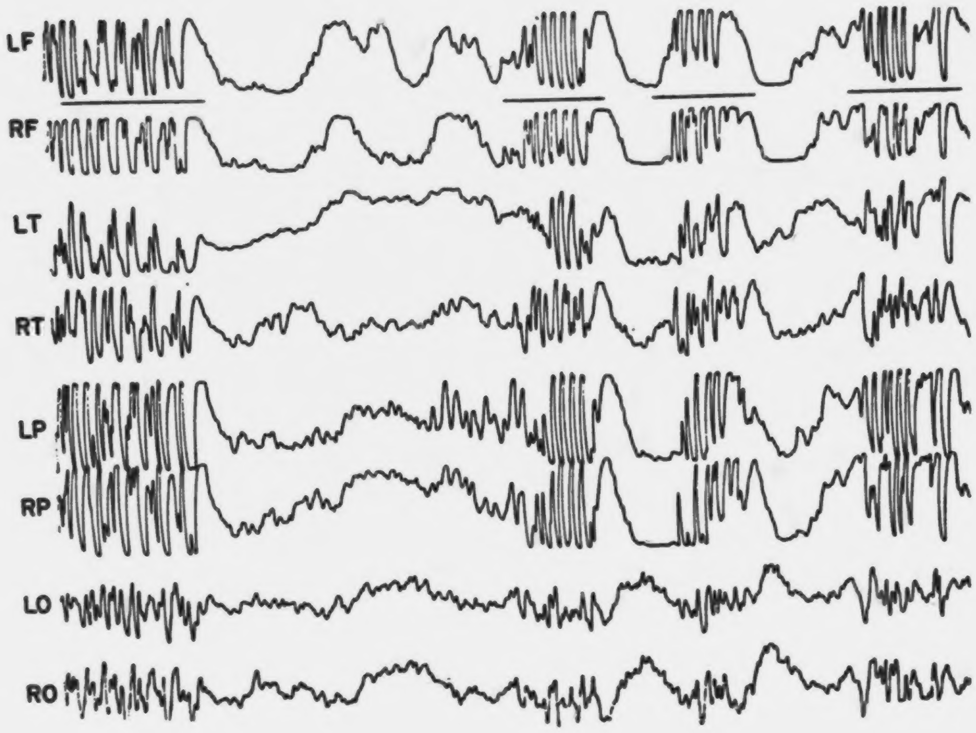

FIGURE 5. Example of multiple spike discharges with simultaneous onset in all leads. These discharges were not associated with clinical seizure activity, but were recorded from a patient who had onset of tonic clonic grand mal seizures at the age of 17. Letters on left indicate areas from which activity is recorded. L = left, R = right, F = frontal, T = temporal, P = parietal, O = occipital. Ear as common reference point.

Penfield places automatism in two categories. In the centrencephalic group under psychomotor automatism, he includes phenomena which are initiated by initial unconsciousness and arrest of activity. Clinically this phase is very similar to a petit mal seizure. It is often followed, in the development of the psychomotor seizure, by licking and chewing movements. These phases are brief and may be unobserved by others, unless in a face to face situation with the patient.

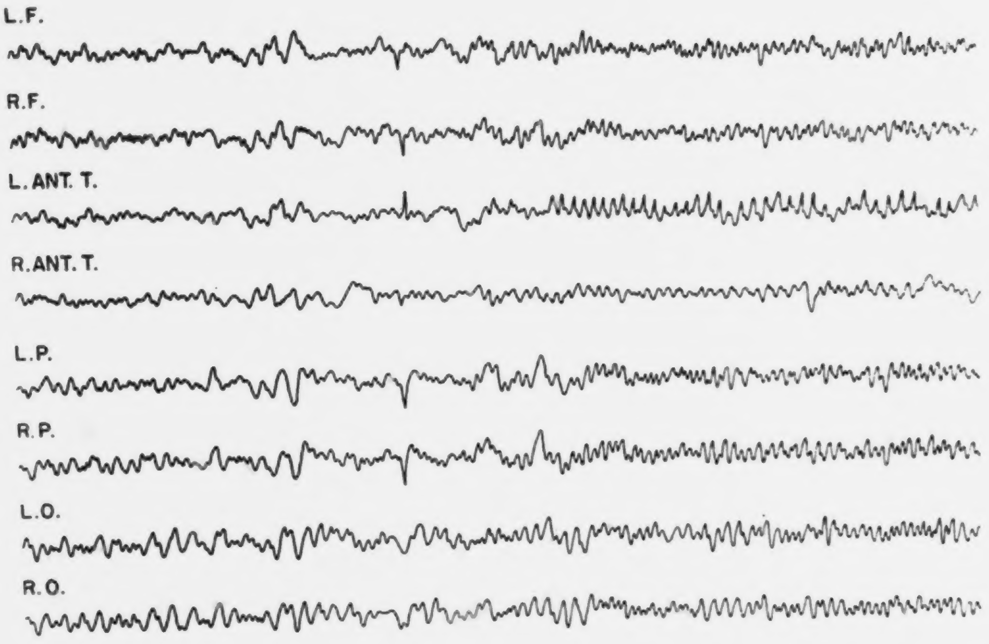

FIGURE 6. Example of EEG of a 51 year old patient with onset of psychomotor seizures at the age of 21. Note the spike discharge followed by a series of spike discharges in the left anterior temporal region. Patient's psychomotor seizures consist of an aura of jamais vu followed by pallor, chewing, fumbling, repetitive vocal utterances, and confusion.

There are other seizure phenomena which reflect dysfunction of the temporal lobe. Among these, and perhaps most interesting from a psychological point of view, are the psychic phenomena. These range from dreamy states to full fledged panoramic visual hallucinations. Feelings of familiarity or unfamiliarity often accompany these states. Intense fear and thought repetition ("It's as if a record got stuck in my head") have also been reported as aurae, as well as musical sounds. Sensory aurae, such as an intensely disagreeable taste or smell, are not infrequent aurae to psychomotor seizures.

Grand Mal Seizures

The grand mal seizure is what most people think of when hearing the word epilepsy. It is often referred to as "a typical epileptic fit." It is no more typical than any other seizure, and such a characterization reflects loose thinking about epilepsy. However, it is certainly the most frightening and disruptive type of seizure to the onlooker and certainly puts the patient out of commission for an effective period physically, and often for a long period socially, especially if the seizures recur in public.

The following description is adapted from Lennox's (1960) first volume on epilepsy. With or without any warning or aura, a tonic spasm of the voluntary muscles of the whole body causes the patient to slump or fall, often like a log of wood, to the floor. With the contraction of muscles (tonic stage) the "epileptic cry" may occur, a cry which had been compared to the scream of a distracted peacock. Respiration is suspended (contraction of chest muscles) and the face becomes livid. The patient perspires and drools. Although this phase seems to have a long duration to onlookers, it rarely if ever exceeds a minute. The clonic stage appears with rhythmical movements, at first appearing like a tetanus, and then, as speed of neuronal discharge decreases, becoming a tremor, and finally becoming jerks alternating with relaxation. It is often during this period that the tongue may be chewed. The bladder may be emptied. After the convulsive movements have ceased the patient lies as if utterly spent. The senses are slowly regained. He struggles to understand who and where he is, what has happened, and falls asleep. After minutes or hours his mind is clear, though he usually has no recollection of events immediately preceding the attack or right after it.

All this sounds as though it lasted a long time, and to the onlooker the duration seems interminable. Actually it is relatively brief, usually not more than five minutes. More often than not a person will want to sleep after a grand mal seizure, even up to several hours.

A seizure runs its course and stops. The onlooker has an almost overpowering urge to do something to prevent the movements, as if that would bring the seizure to an end. It will not, and holding on to arms and legs with this purpose in mind can result in injury to the patient.

All that should be done is pulling the patient away from fixtures, removing furniture from his immediate vicinity, getting a pillow under his head if he is on a hard surface, and turning the head when possible so that saliva will run out. If a second seizure follows without the patient's regaining consciousness after the first one, the person should receive immediate medical attention. Repeated seizures without the regaining of consciousness between seizures, known as *status epilepticus,* is a serious and sometimes fatal event.

In a grand mal seizure the entire nervous system—hemispheres and brain stem, autonomic as well as central—is involved. The EEG shows a series of fast (fifteen to forty per second) sharp waves (see Figure 7) which build up in amplitude and are generalized. Following the seizure there is a slowing and flattening, expressing the literal exhaustion of the neurons.

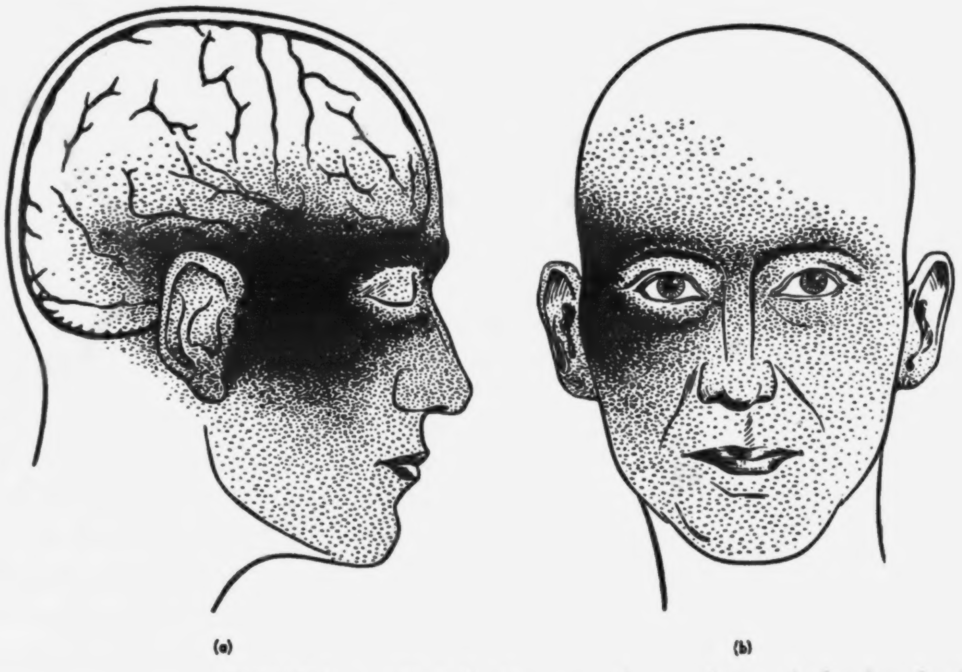

FIGURE 7. Distribution on the surface of the head of focal seizure activity of the psychomotor type (i.e., anterior temporal lobe focus). The voltage of the spikes is indicated by the heaviness of the stippling.

The onset of a grand mal seizure may give a clue to the origin of the discharge which initiates the neuronal events culminating in a convulsion. Obtaining an accurate seizure description can be of prime importance in localizing a brain lesion, such as a tumor, which may be treatable surgically. Eye turning to one side, or adversive eye movements, is usually caused by a lesion in a specific area of the contralateral frontal lobe. Only an arm or leg on one side of the body may be involved initially and successively, usually referred to as a focal motor seizure, or Jacksonian seizure, indicating initial involvement of successive groups of neurons of the motor strip of the contralateral hemisphere. There may be a sensory march of numbness or tingling, implicating the contralateral parietal region. Flashes of light in one half of the visual field indicate involvement of the contralateral occipital lobe. As we have mentioned before, elaborate visual and auditory experiences and dreamy states represent a focus of discharge in the temporal lobes. Aphasic difficulties point to involvement, in the great majority of cases, of the left temporal parietal area.

Grand mal seizures with a focal onset are classified by Penfield as focal cerebral seizures. When the onset is initial unconsciousness, the origin may be in the centrencephalic region. Penfield follows this rationale in including such a grand mal seizure in his centrencephalic group. Lennox classifies any convulsive seizure as part of his convulsive triad.

Grand mal seizures occur at any age. Except for febrile seizures, the earlier the age of onset of major convulsions, the poorer the prognosis. Seizures during the stages of brain development are an important factor in interfering with normal mental growth, whereas they usually do not cause mental deterioration in adults. Repeated seizures early in life apparently reflect, or result in, brain damage, thus interfering with normal development.

Conditions Confused with Epilepsy

1. *Syncope* (simple fainting). Occurs when the person is in an upright position. There is spontaneous recovery in prone position. No motor movements except slumping to the floor.

2. *Narcolepsy.* Recurrent sudden involuntary sleep attacks, especially upon relaxation.
3. *Cataplexy.* Recurrent sudden losses of muscle tone in which the person simply folds up. Precipitated by sudden emotion, such as laughter, or need for sudden quick action. Often associated with narcolepsy. No unconsciousness.
4. *Sleep Paralysis.* On waking the person finds he cannot move a muscle. Typically, lasts only a few seconds. No unconsciousness.
5. *Fugue State.* This is a condition during which the person does not appear to be unconscious, but for which he has amnesia. When the state is long lasting, e.g., several days, it is rarely an epileptic state. The person in a dissociative or hysterical fugue state rarely injures himself or emerges the worse for wear. Typically he shows amazing lack of concern for how he may behave. Occasionally an epileptic may have a clouded state of some duration, which is difficult to distinguish from an hysterical fugue state. He usually emerges much the worse for wear and is concerned about what has taken place.
6. *Hysterical Seizure.* Motor disturbance is usually in the form of stiffening with arching of the body, or incoordinated thrashing about. A frankly sexual flavor is sometimes easily identifiable. There is no personal injury incurred. There is never urination or tongue biting. A person who has only hysterical seizures rarely has one when there is not a good chance of being observed.

Sociopsychological Factors

So far we have discussed some of the more common forms of seizures and some of the intricacies of neurophysiological correlates. The importance of a good seizure description, including subjective experiences as an integral part of the seizure, in arriving at an accurate diagnosis, has been emphasized.

A knowledge of these events should also lead to a better understanding of the individual patient. The classification of a patient as epileptic can tell you something about gross symptomatology, but it communicates very little else. The use of the word epilepsy as a unitary concept has produced a sociopsychological climate which is not only unreal but also disastrous for many people with epilepsy. The following sections point out some of the consequences of this climate.

Epilepsy and the Law. Many of our laws regulating the actions of people with epilepsy are anachronistic. They reflect a cultural lag more related to superstitions about epilepsy than to current realities of medical control and knowledge about the epilepsies.

Roscoe Barrow, Dean of the University of Cincinnati College of Law, and Howard Fabing, a neurologist with particular interest in epilepsy, investigated the laws applying to epilepsy in this country at the request of the American League Against Epilepsy (1956). Their investigation was made between 1954 and 1956.

Driver's License. Many people raise their hands in horror at the thought of a person with a history of seizures driving a car. There are physicians who categorically tell patients who have had seizures that they should never drive. This is a very easy way out for the physician. However, with adequate medical control, is this necessary? What about the person who has seizures only in his sleep? What about the person who has a long warning or aura? Not having a driver's license in this day and age can create severe difficulties in getting to a job. It is often viewed by a person long free of seizures as proof of what he often has believed in the first place: "I'm an epileptic and therefore not eligible to do much of anything." Denial of a license when seizures are admitted drives the seizure patient underground. He denies he has seizures, does not get adequate medical treatment, and gets the license anyway.

It is certainly appropriate for licensing laws to take account of seizures as a hazard to driving. The way this is done varies from state to state. Four states provide for automatic denial of a driver's license to epileptics. In 32 states epilepsy is not mentioned but, in practice, is included as one of the physical handicaps which may be great enough to prevent the driver's exercising ordinary and reasonable control over a motor vehicle. Only four of these states have statutes which require medical information to enter into such a judgment and there are no guarantees as to what weight such information will have in the administrator's decision.

The Wisconsin statute is cited as a model which works well. It contains guarantees for traffic safety as well as for the rights of people with epilepsy. It stipulates general conditions which permit the administrator to issue a license and provides for a right of appeal when the administrator denies it. The general conditions include certification by a physician that the applicant is under treatment and free of seizures. The length of the seizure free period is not stipulated by statute, but a two year period was initially used. Under these conditions a regular license is issued. If the license is denied, the applicant may appeal for a review by a board made up of two physicians, qualified in the diagnosis and treatment of epilepsy, and a licensing official. The determination by this board is binding on the administrator. If the board decides for the issuance of a license, the applicant is issued a limited permit for six months, at which time it is renewable on the statement by the physician that the applicant is still under treatment and has been free of seizures.

The number of epileptics applying for driver's licenses has increased in Wisconsin. It is gratifying to note that of 280 applicants granted a regular license, none had been revoked. Of the 84 granted a limited license, four had had their licenses withdrawn: two of these were for operating a motor vehicle while intoxicated rather than for recurrence of seizures.

Employment. Difficulty in obtaining or keeping employment is a chronic complaint of epileptics. Employers' statements to patients are that their insurance rates will go up; insurance companies deny this. Employers actually believe that accident rates will go up. Questionnaires reveal that many employers think that epileptics are mentally inferior; thus their fears of increased accident rates are not entirely based on seizure occurrence.

A study by the US Department of Labor in 1950 indicated the rate of nondisabling injuries to be four in a group of nonepileptics and 5.5 in a group of epileptics; the frequency of disabling injuries, 7.6 and 8.3, respectively. This was equivalent to less than one injury per million exposure hours. The time lost rate was less for the epileptic group: .02 days per hundred scheduled working days, as compared to .13 days lost for the nonepileptic group. Despite these facts there is still strong resistance to employing a person with a history of seizures. An obvious result is that many epileptics stop looking for work or deny their seizure history and often fail to obtain appropriate job placement.

One approach to the problem is the waiver of compensation for an injury caused by a preexisting impairment. Practically, however, it is often extremely difficult to establish whether or not an accident is caused by a seizure. The waiver, furthermore, violates the spirit of the Workmen's Compensation Law.

Another approach is the Second Injury Fund. The procedure involving payment from such a fund substantially increased the employment of individuals handicapped by loss of limb, and it was hoped that all prior existing conditions which employers considered as handicaps to employment would be covered. However, latent or nonvisible impairments were not covered, and the establishment of these funds has not helped the epileptic, cardiac, diabetic, or recovered TB patient.

In 1955 Ohio passed a law to cover so called latent disabilities within the Second Injury Fund. The latent disabilities covered were derived from a study of disorders named by placement agencies which actually created difficulties in placing people in jobs. Epilepsy was one of twelve such disorders. Unfortunately it applies only in cases ruled as permanent disability. Illinois has more recently introduced a law providing broader coverage.

In setting up provisions to improve the employability of the epileptic there are admittedly many provisions which should be thought of. Two primary ones are: (a) employers are in business to make money, not to run a charity; and (b) epilepsy is considered by most employers to cut down on the profits. The prospective employee who is subject to seizures should also be aware of this. Too often his feeling sorry for himself leads him to expect the employer to give him a job out of pity. He should avoid saying "I have epilepsy." Instead he should make it clear that he has a particular type of seizure, that these occur only so often, and that he is under modern medical treatment for this disorder.

He thus gives the impression that he is able to function adequately in spite of his disability. He should be familiar with the statistics which indicate that controlled epilepsy does not increase accident rates and absenteeism. Emphasis on what he can do and how he is qualified for the job he is applying for can do much to destroy the stereotype of the epileptic and can promote his chances of getting a job on his merits as a qualified individual.

Marriage and Eugenics. The idea that epilepsy is a hereditary and degenerative disease is strongly reflected in laws dealing with marriage. Several states, as late as 1958, had statutes prohibiting marriage of epileptics, and classified them in these laws with habitual criminals, the mentally retarded, and the mentally ill. Some of them make an exception if the woman is beyond child bearing age.

The real emphasis here is on the assumed degenerative nature of the disorders. Other defects in which the hereditary mechanism is much better known and more clear cut than in epilepsy, such as hemophilia and Huntington's Chorea, are not mentioned in marriage laws of any state to my knowledge.

I think it is now well established by many studies that epileptics as a group are not feebleminded, do not deteriorate, and do not become psychotic. But the laws were made when the common view of epilepsy was, and I quote from McCurdy's (1916) monograph, "One of the most typical features of the disease is the mental deterioration which so often accompanies the disease, so frequently that some epileptologists include it in the definition [p. 2]."

A number of states still express this view in their marriage laws. In such states it is a crime for a person who has seizures to marry, for a person to marry another who has seizures, or for anyone to issue a marriage license to such persons or perform the marriage ceremony itself. This is an infringement on civil liberties as great as that imposed by race. A simple maneuver is to strike the word epilepsy from such laws. Three states, Wisconsin, Connecticut, and Kansas, have done this as late as 1955.

In 1927, the Supreme Court established the constitutionality of sterilization. In 1958 there were sterilization laws in 28 states. Seventeen of these had laws specifically applicable to epileptics: in 14 the laws are limited to institutionalized epileptics. In actual practice, apparently, not much is done because of recognition of the genetic unsoundness and the difficulty in enforcement. But the laws are still on the books and can only contribute to the stigma associated with epilepsy and reinforce outmoded beliefs.

Psychological Aspects. I want to discuss now some psychological problems that the person with epilepsy develops in structuring the perception of himself as an epileptic, some factors which contribute to this self image, and some general ways of dealing with these perceptions which may lead to more effective adjustment.

Epilepsy is unique in that the nature of the disorder itself permits little firm or consistent perceptual structure. The observable manifestation of the disorder, the seizure, appears unpredictably, sporadically, and suddenly, lasting only moments. Yet, while it lasts, it is completely disabling to the patient and frightening to the onlooker. Between these episodes neither the patient nor his associates have any constant cues for judging whether he is sick. A second factor which obscures a clear perception of his illness is that the patient, because of unconsciousness and amnesia, does not experience himself at the height of his disorder. What he may be aware of is (a) an aura; (b) his being in a different place or position, sometimes sore and exhausted; and (c) the reactions, expressions, and behavior of those around him. Often the faces look horrified. More often than not people refuse to tell him what happened, what he actually did while he was ill. It is small wonder that many patients become somewhat paranoid under these conditions.

The only constant factor in the perception of epilepsy is a cultural loading of superstition. To most people, including the patient, epilepsy has connotations of falling to pieces mentally and physically. Any illness is likely to have an effect on the self image. People who develop seizures appreciably shift their perception of self in the direction of decreased competence and experience a considerable threat to their personal integrity. Too often this shift is reinforced by important life figures at home, work, or school.

Most people are uncomfortable and frustrated when attempting to make plans for living with a situation which may change drastically and unpredictably from day to day. Most of us attempt to work out some method which will reduce this discomfort and anxiety. Escape from the situation is not possible for the epileptic, although it is open to his associates. (Is this what is taking place in not employing an epileptic, in not keeping an epileptic child in regular classes, in institutionalizing epileptics?) For many patients and their families the solution for the alleviation of this ambiguity is very frequently that of unrealistic structuring of the perception of the illness around one of two poles. "I (or he) am really a sick person and should be treated like a sick person all the time" is one pole. The other is, "I am not really sick, and if I ignore it, it will just go away."

The patient who develops the first attitude—"I am an epileptic and therefore very sick"—may become overcompulsive about medical and dietary regimes and about what he should and should not do. He often becomes demanding and dependent. There is an insidious development of living for his illness. The parent who takes this way out in dealing with a child's illness wreaks all the ills of overprotection. The child never develops attitudes of self reliance and may grow up with the attitude that his seizures are the thing that count most in his relating to others.

The second attitude of denial of illness often leads to neglect in taking medication and to refusal to take account of one's disability in driving and work. Overcompensation for felt, but denied, disability is apt to develop and the person may become hostile and aggressive. Relatives who deny epilepsy often insist that the patient is "putting on," that he is mentally ill. The patient, probably influenced by the superstitions he, too, has grown up with, is often quite willing to believe there is something mentally wrong with him, and often delays seeking appropriate medical treatment.

The extremes of personality characteristics mentioned above have been considered to be part of an epileptic personality, probably present premorbidly, and not likely to be subject to change. Although premorbid personality traits certainly predispose a person's reaction to catastrophic events, I am proposing here that the so called epileptic personality is in large part a reaction to repeated seizures in our social milieu, and represents an attempt to adapt to an ambiguous perceptual situation. Changing the perceptions should lead, if the reactive concept holds, to more healthy ways of the patients' dealing with their illness.

At the Boston VA Hospital, several of us worked with groups of epileptics on a group therapy or group discussion basis. As a result of this work we became convinced that in a substantial number of epileptics, perceptions of self as epileptic could be shifted drastically, and patients could be led to more adaptive ways of dealing with their illness. After many false starts, the general approach that seemed to work best was to eradicate the use of epilepsy as a disease entity and to focus on the realistic limitations of the individual variety of seizures on the one hand, and on the other hand, focus on what the person is, beyond being a person with seizures. In a group setting, with the leader constantly reorienting the discussion around the patients as individuals, they are able to shift their attitudes and help group members to do the same. They leave the hospital much better equipped to live realistically with their illness rather than for it or in disregard of it.

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Organic Language Disorders in Children

KATHRYN BARTH HORTON

Language has been described as that characteristic which makes us human, for it serves as a primary characteristic differentiating us from our predecessors along the road of evolutionary development. Language is, of course, our primary tool of communication and semantic transfer. Thus the role of language is pervasive in the human being's ability to adapt, for it represents the major vehicle through which we organize our world and achieve some degree of system out of the chaos of the infinite number of sensory impressions that we take in during a lifetime. Language facility then assists in man's capacity to think and his ability to act freely. If for any reason, organic or nonorganic, the acquisition of language is interfered with, total development is altered. It is difficult, therefore, to describe language as simply being one of a number of component parts comprising human behavior. Rather, language should be thought of as an all pervasive function in any analysis of behavior.

If we think about the implications of disordered language in a child, we soon recognize the fact that difficulties in the use and/or comprehension of verbal symbols represent a major problem comprising the totality of learning disabilities. Kirk (1963) emphasizes this point strongly. We shall devote our attention here specifically to those learning disabilities that are primarily linguistic in nature. The term most applicable in describing or labeling these problems is childhood aphasia. The numerous writings on this subject make it evident that a substantial body of interested specialists representing a broad range of habilitative and educational services believes that there exists a special entity of impairment in the language function, especially in the learning of language by young children (West, 1962). It is generally agreed that aphasia in childhood is the result of cerebral dysfunction which is either congenital or acquired, and that aphasia is characterized by both linguistic and nonverbal behavioral manifestations. Osgood (1963) defines aphasia as a "nonfunctional impairment in the reception, manipulation, and/or expression of symbolic content whose basis is to be found in organic damage to relatively central brain structures [p. 8]." He makes a further differentiation between congenital aphasia and childhood aphasia. Congenital aphasia is defined as "a condition in which either poor endowment or brain injury occurring before, during, or after birth prevents the child from acquiring language [p. 21]." Childhood aphasia, on the other hand, is described as "language impairment occurring after language has been acquired in the normal manner [p. 21]." This differentiation is not commonly made in the field of speech pathology and audiology since the majority of problems observed in children are those that Osgood would describe as congenital. Congenital aphasia and childhood aphasia are used in the main as interchangeable terms. It is generally agreed, however, that it is important to exclude in the definition of aphasia in children language or linguistic problems that are associated primarily with the following conditions: (a) intellectual deficiency, (b) sensory impairment (primarily deficits in hearing), (c) central nervous system damage resulting in physiologic deficits of the peripheral speech mechanism, (d) severe emotional disturbance, and (e) delayed maturation in language development resulting from environmental, physiologic, or emotional factors that are not primarily due to central nervous system impairment.

A variety of terms is used to denote aphasia in children. These terms reflect variations in the point of view of the persons utilizing the terms; that is, they are different in that they indicate a different component of the total syndrome being emphasized or reflect a difference in the diagnostic or habilitative point of view. This wide variety of terminology includes, in addition to childhood aphasia and congenital aphasia mentioned above, such terms as motor aphasia, expressive aphasia, receptive aphasia, global aphasia, congenital word deafness, central auditory imperception, central deafness, congenital verbal auditory imperception, agraphia, alexia, apraxia, developmental language retardation, and specific language disability. Such an array of terminology suggests that we are speaking of numerous conditions rather than a single condition—that organic disturbances in children present a clinical picture with many aspects. Benton (1963) makes this point and suggests that we may be dealing with a number of essentially different conditions which have as their only commonality the fact that there is a failure of or difficulty in the acquisition of language skills.

Definitions of these labels and interpretation of their appropriate usage can be facilitated by viewing them within the context of a language model. Wepman (1960) proposes a three part system, comprising (a) an input system which is specific to sense modalities, (b) a central integrative system which does not imply modality specificity, and (c) an output system which is also specific to modalities. Disturbances of input are described by Wepman as agnosias; the type of agnosia is further defined by the sense modality involved. Therefore, for the purposes of language learning, we may speak of disturbances of input in the auditory, visual, or tactile senses or of combinations of problems in any two or all three of these channels of intake. Disturbances of output are referred to as apraxias and include several modalities of expressive potential. Agnosias and apraxias can be considered as disturbances of decoding and encoding, respectively and may involve verbal and/or nonverbal functions. Disturbances of the central integrative system are described as aphasias. Aphasias reflect disorders in the retention, retrieval, and expression of verbal symbols and have major intersensory significance. Learning disorders that involve primarily language dysfunction include the aphasias or dysphasias, the alexias or dyslexias, and the agraphias or dysgraphias. In addition, agnosias and apraxias, reflecting dysfunction in either the intake or output of verbal symbols, should be included.

When one considers the relative importance of the various sense modalities available to the human in language learning, it becomes immediately apparent that audition plays the most pervasive role in man's acquisition of language. We learn to talk and understand primarily because we hear. This point is highlighted by considering the normal acquisition of language where it is apparent that high levels of auditory verbal comprehension and verbal expression are achieved prior to the development of reading or writing. The child who is deficient in auditory sensitivity on the other hand will show significant retardation and deficits in his language ability from the earliest stages, both receptively and expressively. This is not to say that he cannot learn language through other sense modalities, but his learning of language through other than auditory channels is necessarily deferred until he has achieved sufficient maturity to use vision for verbal learning. The impact of the deferring of language learning has been discussed by a number of persons including Simon (1957) who posits that there are critical neurologic periods for language learning. The implication is that delay in the learning of language irrespective of etiology reduces the potential for learning. Even during the first year of life, highly reinforced auditory kinesthetic patterns are learned, to be reflected in the second year of life in the utterances which are the words of the young child. Hardy (1962) points up the importance of audition to language learning in his discussion of the concept of "auding" which he describes as the process necessary to the management of acoustic information. In auding, the human is processing information that he takes in auditorially, organizing it into patterns, and storing it for later retrieval. Hardy draws an analogy between the process of auding and its relation to hearing and the process of reading which is a function of the visual modality. Disturbances of auding or auditory processing are consistently observable in aphasic disorders.

The firmest basis for any system or schema undergirding evaluation of organic language disorders is a model of language. Such models are discussed in the work of Wepman (1960), Blair (1963), and Kirk and McCarthy (1961). These models imply specific diagnostic and evaluative procedures to define the parameters of the language disorder. Implicit in the definition of the parameters of the disorder are the dimensions that suggest therapeutic and educational guidelines. Generally speaking, assessment procedures are oriented toward either classification or diagnosis. In the former, the vehicles of evaluation are group type or categorical tests which point to an appropriate category in which to classify the person and his problem. Tests such as these yield global scores as are represented in typical IQ or MA scores. On the other hand, the goal of diagnostic testing is the detection and thorough definition of the person's specific abilities and disabilities. This approach does not rely on global scores but rather yields profiles of functioning which directly suggest remedial or therapeutic programing. Useful and functional evaluative procedures for organic disorders must adopt this diagnostic approach. The Illinois Test of Psycholinguistic Abilities was developed for specific application to children with this intent in mind. In addition, Bangs (1961) suggests a broader procedure which incorporates a variety of tasks drawn from existing tests.

These and other proposed evaluative formats for testing organic language disorders in children all imply that adequate assessment must be modality oriented. In testing the integrity of sense modalities, assessment is suggested at two levels—definition of the intactness of a specific sense modality functioning in relative isolation and assessment at the level of intermodality function. A suggested sequence of levels of assessment within any one sense modality going from simple to complex functions is (a) sensation, (b) attention, (c) recognition, (d) retention, (e) discrimination, (f) imagery, (g) recognition of the verbal symbol, and (h) retrieval and use of the verbal symbol. This sequence is based on the sequence of levels of function required for the learning of a verbal task. The allusion that there is nonequivalence between sense modalities with reference to their potential for learning has been made frequently. Writers have suggested that individuals vary from each other with reference to their auditory and visual potentiality for learning. Such thinking has resulted in the use of terms such as "audile" and "visile" to point up an individual's most fruitful channel for learning. If this point of view has merit, it bears distinct relevance to language learning and would imply that assessment of language disorders concerns itself with the definition of modality strengths and weaknesses.

It has been suggested previously in this paper that analysis of large groups of language impaired children reveals the presence of widespread deficits in the area of auditory functioning. Boshes and Myklebust (1964) comment on the general difficulty encountered in auditory blending tasks by their patients with either suspected or known dysfunctioning of the central nervous system. Hardy (1962) and Blair (1963) both comment on and support the notion that auditory abilities seem to be primarily impaired in aphasic disorders. These findings support the notion that the assessment of auditory integrity comprises one of the largest components of an evaluative scheme for organic language disorders. Myklebust (1963) suggests the inclusion of the following types of tests in a language battery: (a) immediate recall of digits; (b) immediate recall of words; (c) immediate recall of connected language in sentence form; (d) immediate recall of series of syllables thereby tapping memory span without including semantic and syntactic clues; (e) ability to synthesize a word from disconnected presentation of the phonemic elements of the word in their proper order; (f) ability to formulate a sentence from a series of words presented at a rate of one per second (Myklebust refers to this as a test of auditory association, tapping the individual's ability in language structure and syntax); (g) immediate memory for rhythm; (h) carrying out three commissions (similar to Binet items) involving the ability to comprehend and sequentialize in time and space a number of acts; and (i) test of auditory discrimination concerned with differentiating very similar phonemic elements.

The role of intellectual assessment is of paramount importance to the diagnosis of aphasic disorders. By definition, an aphasic problem implies depression of linguistic functions with abilities in areas of behavior that do not require verbal skills approximating those expected for the chronological age of the individual. Therefore, the prototype of the aphasic child would show intelligence levels on performance types of tests that approximate his chronological age. There has been disagreement within the field concerning whether or not language disorders should be considered a type of mental retardation. It certainly would be fallacious to exclude language from our construct of intelligence and adopt the point of view that aphasic children are as intelligent as normal children. Certainly one cannot deny that language comprises one of the most, if not the most, important component of what we call intelligence. However, it must be emphasized that disorders of aphasia imply near normal levels of functioning in nonverbal areas. Therefore, intellectual assessment that provides for a comparison of verbal and nonverbal abilities is desirable as a part of the evaluation scheme for organic language disorders. The Wechsler Intelligence Scale for Children (WISC) is probably the most adaptable single instrument for this purpose. The downward extension of the WISC, the Wechsler Preschool and Primary Scale of Intelligence (WPPSI) is heartily welcomed, since there are relatively few available scales for the preschool child allowing a comparison of verbal versus performance abilities.

Another point in this connection should be stressed. Many of the children that we would classify as aphasics present a multiplicity of problems that often include some degree of mental retardation. Therefore, a practical clinical approach suggests that we not attempt to dichotomize completely the aphasic and mentally retarded child, rather that we think of aphasic children as having various degrees of mental retardation and of the typically retarded child as having no degree or various degrees of aphasia. In this connection, the term "aphasoid" has been offered to describe the overlay of aphasic like behavior that may be a part of other syndromes.

The role of language in the development of intelligence is a fascinating topic to consider for it has been frequently observed in evaluating very young children with suspected language dysfunction that their apparent intelligence, as defined by nonverbal tests, appears to be relatively intact and in some cases well above expectation for their chronological age. However, this estimate of their basic capacity to learn things other than verbal may be deceptive with reference to its temporal reliability; this fact becomes increasingly apparent as the child gets older. As language takes on increased importance as a parameter of what we call intelligence, these children appear and test more and more like mentally retarded children. An illustration of this very point is evident in one of the cases which is presented later.

One of the premises by which we define an organic language disorder is based upon neurologic dysfunction within the central nervous system. The dysfunction in language is simply one reflection of a lack of integrity of the central nervous system. Another reflection, as is suggested in the preceding section of the paper, may be a deficit in overall intellectual capacity. Other components of the syndrome are well known and very probably will be discussed at length in later chapters. These characteristics represent the behavioral and perceptual disturbances that are evident as a part of central nervous system pathology. In examining a group of language disordered children, one will observe many of them to be hyperactive, catastrophic, disinhibited, perseverative. In fact, all of the characteristics which are usually considered to be part of the syndrome of brain injury will probably be observable to some degree. It has been noted by a number of clinicians that these disruptions in behavior seem related to the degree of disturbance of the auditory function. That is, these manifestations of lack of behavioral integrity are less in evidence in language disordered children where the visual function shows greater impairment than the auditory function (e.g., dyslexic children). All of the criteria applicable to the testing and educational and therapeutic management of brain injured children are certainly equally applicable to the management of aphasic children.

To extend further this discussion of the disruption of behavioral integrity in children with CNS dysfunction, we must give some attention to the nonspeaking child who, in all of his behavior, appears to be autistic. In our tendency to classify problems into mutually exclusive categories, we sometimes fall into the trap of thinking of a child as being either normal, mentally retarded, sensory deprived, aphasic, or autistic. This indeed is a trap, for the nervous system itself shows no such categorical division, and we should, therefore, not expect to see neat diagnostic differentiation in the children with whom we work. Language and speech clinicians all too frequently are challenged by youngsters who appear as autistic in their behavior yet who also seem very aphasic. Referral to the psychiatric specialist many times results in management purely from a psychiatric point of view. The assumption implied is that, when the child's psychological status has shown significant improvement, he will then communicate in a more proficient manner via language with his world. Unfortunately, this result has not been frequently observed. It is probably much safer to suppose that dysfunction of the central nervous system may reflect itself both in disturbed behavior as is evidenced in autistic children and a disturbed capacity to learn language. Further, it is probable that these two disturbances of function interact and complicate each other in terms of the observed symptomatology from very early developmental levels. Therefore, I believe it behooves us to evaluate the language learning potential of the speechless child with autistic appearing behavior as extensively as our instruments and clinical insight permit and the child will tolerate. A child typifying this kind of complex disability is presented in one of the later case studies.

Two very opposing philosophies are evident in the remediation of language disorders in children. Probably the most prevalent one adopts the point of view that those areas of behavior basic to language learning that reflect deficit should be strengthened. This point of view holds that we should strive toward reaching equivalent levels of ability for all facets of behavior relative to the acquisition of language. Therefore, according to this philosophy, therapeutic and remedial procedures should concern themselves with strengthening the weaknesses and ultimately in achieving a leveling. The opposing point of view, by contrast, would emphasize capitalizing on the assets or strengths of the individual that are important to language acquisition and would advocate the direct teaching of language utilizing the more intact channels for learning. This latter point of view would have as its basis the thought that the most important goal is the learning of language; the means by which it is learned is secondary. Myklebust (1963) adopts the former position and stresses the necessity of preventing overlearning in the most intact area, thereby suggesting achieving the maximum level of function in the area of greatest deficit. He places great stress on the principle of simultaneity with reference to sensory stimulation. In studying this principle, Myklebust is now experimenting with computer programed language learning.

Mildred McGinnis (1963) was the originator of the McGinnis Association Technique for teaching language to aphasic children. This approach has been applied at the Central Institute for the Deaf, a well known and respected institution for the education of deaf and aphasic children, and has received wide acclaim. Essentially, the McGinnis approach stresses concurrent teaching utilizing all sensory modalities and thus stresses intersensory facilitation. Miss McGinnis, in her teaching technique for aphasic children, establishes strong auditory-kinesthetic-visual associations. Her approach is generally analytical and stresses the teaching of the articulation of speech concurrent with the recognition of the visual symbol representing the speech unit concurrent with the writing of this speech unit.

The Initial Teaching Alphabet (ita), introduced in England by Sir James Pitman, offers an interesting approach to the teaching of language to language disordered children. This alphabet consists of a repertoire of 44 visual symbols, each representing a single phonemic element and each bearing a strong similarity of form, especially in the upper half of the symbol, to traditional orthography. One of the problems encountered by aphasic children is the lack of consistency in relating phonemic events to the visual symbols used in reading. For instance, one sound can be spelled in a variety of ways traditionally. The sound common in the word "I" has 22 different spellings; therefore, the language disordered child is challenged to learn as many as 22 alternative visual referents for this single phonemic event. This task represents an arduous one for children of normal language capability. The challenge that the aphasic child faces is sometimes an overwhelming one. Therefore, the ita offers a potentially valuable tool by which reading could be taught to language disordered children. Its application to the McGinnis Association Technique is worthy of investigation. Researchers at the Bill Wilkerson Center are presently studying the application of this alphabet to speech and language disordered children.

I would like now to summarize some of my own philosophy concerning the management of language disordered children. There is rather general professional agreement that remediation demands an inclusive educational program in which emphasis is placed not only on teaching the child to talk and to understand speech but also on teaching language via all channels of intake and as an integral approach to other academic subjects. Thus, those of us interested from both the therapeutic and the educational points of view are challenged to blend our talents in planning and implementing programs for children with language problems.

Second, successful remedial programs for children with organic language disorders demand identification and implementation of educational and therapeutic management at early preschool levels. The fact that a child does not have language with which to communicate his feelings, needs, and thoughts makes him vulnerable to the development of pervasive psychological problems. In deferring treatment for aphasic children until age six (the age typical for school placement), we are neglecting the most fruitful period in his developmental sequence in which to assist him in the acquisition of language. Therefore, aphasic children must be identified early, and they must receive intensive treatment early. The areas of greatest capacity should be strengthened, and language should be taught early, irrespective of the modality used to teach it.

A third point is that organic language disorders do not represent a homogeneous group of learning disorders. The implications of this fact with reference to remediation and education are self evident. Aphasic children have many commonalities, but they differ markedly from each other in terms of available and appropriate inroads to educational treatment. In my clinical experience with aphasic children, I have seen as much variation between two aphasic children as existed between either one of them and a normal child. Therefore, we should prepare ourselves for flexible and multiple educational approaches for language disordered children. In connection with this point is the contention that education of the language disordered child must be modality oriented just as assessment is modality oriented. Specific programs of remediation must stress modality strengths rather than devote total emphasis to work on modality weakness.

Organic language disorders in children cannot be easily separated from the general concepts of mental retardation. Indeed, aphasic children pose as heterogenous a group of educational problems as do mentally retarded children. Greater specialization of our educational programs for the mentally retarded child might ultimately satisfy the educational needs for the aphasic child. Increased specialization of programs for the retarded requires an expansion of, or in some cases a different approach to, educational evaluation. To my knowledge, evaluating the educational needs of the retarded child has focused on assessment of the degree of retardation, and thus educational programs have been developed around guidelines suggested by IQ ranges. I believe, however, that the heterogeneity of mental retardation calls for a different approach in educational evaluation. It might be worthwhile to consider thinking in terms of a more descriptive classification of educational needs, not necessarily based upon the degree of overall intellectual deficit. Tests that profile specific abilities and disabilities, as exemplified in the approach of the ITPA, would seem to offer potential for analyzing and pointing up the educational inroads that are appropriate for a group of retarded children irrespective of their IQ score. If such a diagnostic educational approach were adopted, it is entirely feasible that language disordered children could, at least in part, be included in educational programing for retarded children. If this approach is not feasible, the approach may have to be based on the development of programs that are specific to perceptual and language disturbances as contrasted to mental retardation.

Following are several cases that represent some of the variety of aphasic or language disorders that one might encounter in a clinical situation. (Case data follow text.)

Case Summary No. 1 is illustrative of a child with aphasia complicated by significant retardation. This child's intellectual functioning consistently showed depression; however, the depth of the depression is more apparent in her later years. Speech ability and expressive language were always affected to a marked degree. This child shows congenital aphasia with more expressive disability than receptive. Her expressive language functioning is significantly depressed in comparison with all estimates of overall intellectual ability in spite of the fact that there is evident mental retardation on all test performances. Educationally, she must be handled as a retarded individual; however, in addition to her formal educational management, speech and language therapy are necessary. In addition, an approach to reading which is consistent with her language and speech learning needs is called for. Possibly the early use of the ita symbols or a similar system in combination with a McGinnis like approach would have yielded much greater development of expressive language.

Case No. 2 represents a problem in which there is a major disruption in the intake of auditory stimuli, specifically auditory verbal stimuli. The diagnosis most appropriate for this child would be an auditory verbal agnosia. In spite of the fact that there is consistent test evidence to demonstrate normal sensitivity to sound, this boy has no functional ability to utilize audition as a channel for learning. From an educational viewpoint, he is essentially a deaf child. However, his handicap is more pervasive than one might expect with peripheral deafness in that even when he hears he has no capacity to associate auditory events and their referents. Thus, language for him must be something other than aural and oral language. This boy responded rather well to the learning of manual language, and if he had chosen to stay in an educational setting for the deaf, his language development might have achieved a much higher level of function. His case represents, I believe, the need to extend beyond our etiologic diagnostic categories for the purposes of educational prognostication. For this is a boy with normal sensitivity to sound who has no capacity to utilize sound for learning and, therefore, must necessarily be educated as a manually oriented deaf child.

Case No. 3 represents the combination of a language disorder and autistic behavior. Efforts to teach her through auditory channels and to achieve some level of oral communication proved fruitless. However, when this child was given an opportunity to learn language manually and to express herself through other than oral means, her lack of behavioral integrity lessened. It is prognosticated that she now may make some satisfactory adjustment psychologically. This kind of evidence points up the danger of dichotomizing diagnostic categories as they relate to educational planning.

Case Summary No. 1

1. Identification
   Name: W. B.
   Birthdate: 2/18/49
   Age at first evaluation at Bill Wilkerson Center: 3 years, 1 month
   Referring Cause: Delayed development of speech and suspected mental retardation.
2. History and Background
   Prenatal and Natal: Full term pregnancy complicated only by anemia and some hypothyroidism. Third pregnancy; all children delivered by Caesarean section. Cyanotic 15 to 20 minutes following delivery.

Postnatal: Negative except for mumps at nine months and tonsillectomy and appendectomy at 22 months.

Development: Sat alone at 6 months, walked at 13 months, and toilet trained at three years.

Language, Speech and Hearing: Prelingual vocalizing minimal, with babbling at six months. First word evident at one year. At age three, vocabulary consisted of 12 words used singly; communication achieved by single words and gestures. Response to sound normal; followed simple commands at age three.

Familial: Negative.

3. Testing and Observational Evaluation

Audiological: Normal responses to pure tones, speech and noisemakers presented in free field testing.

Social Maturity: SQ of 94.

Intellectual: Formal testing not possible at three years; however, on selected Binet and Arthur items, she showed greater difficulty with abstract forms than with concrete materials. Could not handle Binet 3 Hole Form Board or the Leiter 2 year level items. At four years, six months, achieved 30 month level.

Speech and Language: Follows simple commands; responds to sounds. Uses speech only when under duress or urged. Has vocabulary of 12 words; uses "jargon" at times. Jargon is primarily vowel utterances; consonants used sparsely and only the early ones are noted when used. Reticent to attempt imitation of speech sounds.

Early Diagnostic Impression; Congenital aphasia. Therapy recommended.

4. Subsequent Findings

At 5 years seen at Cove Schools for four month period where fourth to sixth grade achievement was prognosticated dependent on development of language. Has been recently educated as EMR. No significant gain of expressive vocabulary. Speech is not attempted.

Recent Test Findings: PPVT, IQ = 64 at 14 years, five months; Goodenough Draw-a-Man, MA=5 years, 9 months at 14-2; Leiter Performance Scale yielded MA=7 at 14 years; Gates Primary Reading Test first grade, six months for paragraph reading and second grade, third month for word recognition; Vineland social quotient=61; Chicago Non-Verbal MA=11-6 at 14; WISC Performance IQ=55 at 14; Bender-Gestalt yielded developmental age of 5 at 16 years using Koppitz norms. At 16 years, vocabulary shows some increase, and a few sentences are evident. Hearing normal.

Case Summary No. 2

1. Identification
   Name: E. J.
   Birthdate: 1/8/47
   First Examination: 7/24/51
   Referred by: Parents
   Referring Cause: Lack of speech and possible hearing loss.
2. History and Background
   Prenatal and Natal: Mother had rubella during first three months of pregnancy. Birth premature at 7 months; weight, 4 lbs., 12 oz.; labor eight hours; forcep delivery; head presentation. Cataract on right eye noted.
   Postnatal: Incubator for ten days. No significant deviations in physical development; no severe illnesses or accidents; no convulsions. Immature behavior and failure to develop speech, together with some indication of lack of normal response to sound.
   Medical: Negative except for suspicion of deafness. Congenital cataract on right eye.
   Description of Problem: The child reportedly babbled, but parents did not know when this occurred. The child did not develop meaningful words other than "mama" and "bye bye" and had not developed sentences up to the time of the initial interview. The parents first became aware that a speech problem existed when the child was between 2 and 2½ years old. The child responded to loud sounds, but the response to sound was described as inconsistent.
3. Testing and Observational Evaluation
   Medical: ENT: Negative, but suspect deafness. No other medical examination reported.

Social Maturity: Vineland score on 7/24/51 gave age equivalent 3.1 and social quotient 69; on 10/22/52, age 3.6, SQ 63; on 11/10/54, age 6.8, SQ 87.

Audiological: No formal audiometry on first visit; no response to spoken voice nor to shouts, but reliable responses to noise makers at moderate loudness. GSR on 4/12/56 indicated hearing within normal range at 500-4000, but with test reliability fair and responses inconsistent. Impression in reports is that hearing loss is not primary cause of language disability.

Psychological: Upon initial examination, child obtained an MA of 2 on a visuomotor test, and an MA of 3 on the Binet 3 Hole Form Board. On 10/28/52 he obtained an MA of approximately 4 years on items involving form perception. Failed items involving color perception in all age levels. On 12/7/55 he scored an MA of 6-3, and an IQ of 70 on the Leiter.

Diagnosis: On the initial examination, the following statement was made: "While there is considerable evidence that deafness may be excluded as a cause of his speechlessness, the total picture obtained left considerable doubt as to the major handicap." Further exploration was suggested, particularly in the areas of intellectual functioning, emotional disorder, and defective or delayed visual development.

The reexamination of 10/28/52 contained the impression that the delay in speech acquisition is the result of a mixed aphasia, congenital in origin.

After approximately four years of educational training, a summary on 8/8/57 contained the impression that the child's receptive disabilities appear to be centered in the perception and interpretation of auditory verbal stimuli. It was further observed that his capacities for interpretation of auditory nonverbal, visual, tactile, and kinesthetic cues were essentially intact and that he was well endowed intellectually, although objective test results were not submitted in verification of this. His behavior at that time was more like a deaf child than a child who possessed a brain injury as such.

Therapeutic and Educational Handling: During the period of therapy at the center, the program included efforts to develop prespeech abilities of recognition and discrimination of form and color; encouragement of growth in receptive ability; stimulation of control of vocalization; efforts to improve social maturity. A program designed to develop the various aspects of language ability, including writing and number concepts, as well as efforts to elicit words, was a continuing project. Progress was reported in all activities except the verbal vocabulary. In 1957, the child was recommended for admission to the Tennessee School for the Deaf because of the fact that the training program used for a deaf child appeared to be the educational approach most likely to develop his verbal ability.

4. Subsequent Findings

Later History: Evaluated at the hearing and speech center in July, 1963, at 16 years of age. Response to hearing tests shows normal sensitivity to sound; however, no comprehension of speech. Communication is by means of gestures; cannot lip read. Withdrew from school for deaf after 1½ years there because he was "homesick."

Recent Test Results: Vineland Social Maturity Scale, SQ = 72; Chicago Non-Verbal Examination, MA = 11 years, 2 months; later, MA = 6 years, 9 months; Gates Primary Reading Tests, 2nd grade, 3rd month; California Reading Test, total comprehension at 2nd grade level (highest score on word form, 4.8 grade level; and lowest score on picture association, 1.8 grade level). Presently being taught by homebound teacher. Reading and arithmetic are reported as most difficult subjects. Has made satisfactory adjustment to home in a small town, drives car independently, and has several friends a few years younger than he.

Case Summary No. 3

1. Identification

Name: S. J.
Birthdate: 5/17/53
First Evaluation: 8/3/54

Family: Parents—college graduates; brother—born 9/57.
Referral: At 1 year and 2 months this child was referred by a pediatrician to Vanderbilt University Hospital, with request for GSR testing. Referred to Bill Wilkerson Hearing and Speech Center.

2. History and Background
   Prenatal and Natal: Pregnancy uneventful. Nine months gestation. Weight at birth: 3 pounds and 13 ounces, but infant was mature in appearance.
   Developmental History: Began to sit alone at 12 months, walked at 24 months. Muscle tones reported poor in infancy; gait was termed awkward during early years. Note: At 5½ years pediatrician reported "physical examination entirely within normal limits."
   Description of Problem: Little babbling reported. Parents felt that she did not hear sounds either soft or loud. She did not like to be touched or held.
3. Testing and Observational Evaluation
   Pediatric: This child had careful pediatric observation as an infant; between 1954 and 1959 there were evaluations in VUH Pediatrics Department; in 1960 extensive testing was conducted at Johns Hopkins Hospital.
   Neurological: VUH reports on EEG: 7/8/58 without evidence of focal or generalized cortical dysfunction; on 3/5/59 EEG and skull films within normal limits. Note: A later report from Maryland mentions two tests showing irregular brain waves.
   Audiological: Findings at BWHSC were inconclusive on first visit but she showed a type of auditory behavior not characteristic of really deaf children. She gave definite and overt responses at the following levels:

250 cps	75 dB
500 cps	75 dB
Masking noise	80 dB
Recorded speech and music	80 dB

GSR testing at this center on 3/4/58 and 3/15/59 indicated auditory acuity within normal limits.

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Part 3:
Psychological Diagnosis

The Use of Psychological Tests in Diagnosing Brain Damage in School Children

HOMER B. C. REED, JR.

Current interest in the identification and description of children with brain lesions is largely a reflection of the increasing sensitivity of educators, psychiatrists, pediatricians, and other professional groups to the fact that many school age children display deviant patterns of behavior which cannot be reasonably attributed to the more traditional explanatory concepts such as psychiatric illness, cultural deprivation, or generalized amentia. When traditional explanations fail, new ones are sought and the inconsistency in the meaning of the concept "brain damage" has acted as only the mildest of deterrents in the current trend to invoke the concept as a sufficient explanation for a wide variety of otherwise unexplainable behavior patterns. There is currently much evidence that the concept of brain damage will be used as a basis for legislation and for other forms of social planning and management, and it is therefore necessary that the scientific basis of this concept be spelled out in as much detail as possible. It would be both erroneous and misleading to say that the concept of brain damage has no fixed meaning; it has instead a variety of fixed meanings bearing scant resemblance to one another and the particular meanings employed at any given place or time depends largely on the professional group using the concept. The early work of Strauss and Werner (1941) has given and continues to give a rich observational base to the concept of brain damage. The adjectives hyperactive, impulse ridden, distractible, emotionally labile, and perceptually disordered all apply to the group of children originally identified by Strauss and coworkers as having sustained some insult to brain functions. It is noteworthy, however, that the evidence in these early studies for identifiable neurologic impairment was very meager and thus the concept of brain injury was anchored almost exclusively in observable behavioral data. To the neurologist or to the pediatrician, however, the concept of brain damage has meant something quite different. Within the neurological disciplines, the concept of

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brain damage has been anchored to such data as anamnestic information describing a probable insult to the brain, reflex asymmetry, anomalous findings on such specialized diagnostic procedures as electroencephalography, pneumoencephalography, or angiography, neurosurgical information, and neuropathological information based either on surgical specimens or on autopsy findings. It has become increasingly the job of the psychologist to reconcile these two concepts of brain damage: that is, to understand the problems of the brain injured child as delineated by Strauss (Werner and Strauss, 1941) in terms that are both acceptable and meaningful to neurological scientists. It is further the job of the psychologist to specify on the grounds of his examination the expected course of development for the particular child whom he has examined and the most effective procedures for remediating the behavioral deficits.

If psychology is to make much headway with the problem as stated above, it is crucial that at least the obvious obstacles which lie across the path to solution of the problem be identified as clearly as possible. It is necessary first of all to recognize as indicated above the different meanings which have attached themselves to the concept of brain damage or the "brain injured child." It can be easily granted that both the neurological scientists and educators have successfully identified large groups of children whose problems are poorly understood. The ubiquitousness of the concept of brain damage, however, does little to insure that any sizable number of children will be found who can be securely identified as belonging both to the neurologists' and to the educators' classificatory systems. This, however, is an empirical question, the answer to which must await the conclusion of the relevant investigative efforts. A second obvious barrier to progress that the psychologist will encounter consists of specifying the criteria to be employed in assigning subjects with the label brain damaged. For the educator, the criteria will consist largely of reliable and descriptively valid rating scales used in combination with whatever reasonably objective data can be gathered concerning the child's educational aptitude and achievement. Since the use and understanding of these measures is a routine part of the training of most psychologists, no more will be said about them in this paper. It is in the area of neurological criterion information that psychologists are most deficient since their background of training and work experience is largely unrelated to the particular problems encountered. It is in this problem area that compromises must be made between what is desirable, what is realistic, and what is necessary. Although it might be desirable for psychologists contemplating research efforts in the area of brain dysfunction to be thoroughly trained in all of the branches of the neurological sciences, such a course of action is obviously unrealistic and hopefully not necessary. It is necessary, however, that the psychologist have a basic appreciation of the kinds of problems that a neurologist faces in his efforts to identify brain injured children and the kinds of instruments which a neurologist has at his disposal for gathering the information upon which to base a decision. Few psychologists recognize, for example, that the neurologist cannot specify in advance the information upon which he will rely for making an inference of brain damage or no brain damage. The critical information may come from anamnestic data as in the case of a child with a history of an infectious disease involving the brain accompanied by prolonged hyperthermia, convulsions, or alterations in consciousness. With an entirely negative case history, critical and sufficient information may come from an electroencephalogram displaying a clear slow-wave focus, from clearly aberrant reflect findings, from an examination of the cerebral spinal fluid, or perhaps from a pneumoencephalogram which reveals clearly enlarged or asymmetrical lateral ventricles. Many psychologists fail to understand that one of the basic dicta of modern medicine is, "Thou shalt not harm the patient"; and that therefore a physician may not engage in many kinds of specialized diagnostic studies which, although undoubtedly helping to satisfy one's curiosity about the brain of the patient, do not directly benefit the patient's health or welfare and may conceivably be of some harm. Unless the psychologist knows the kinds of information that the neurologist's tools reveal together with the conditions under which certain kinds of information can be trusted or not trusted, he is in no position to assume responsibility for the execution of the much needed investigative work. The difficulties psychologists encounter in attempting collaborative investigative efforts with neurological scientists could be detailed ad infinitum. These difficulties derive in large measure from psychologists' lack of familiarity with the world of clinical medicine. There is as yet little evidence of awareness of this problem on the part of the major university centers responsible for the professional and scientific training of psychologists. In the absence of any major change in university training programs, psychologists will necessarily have to secure training in the problems and techniques of clinical medicine in whatever ways are open to them. This means for most psychologists, that training will be limited to reading the appropriate text books plus, perhaps, occasional participation in ward rounds, diagnostic case presentations, etc. This kind of informal training can, given an industrious psychologist-student, result in at least the minimal level of sophistication and competence necessary for responsible understanding of cross disciplinary problems. In addition to the above basic knowledge of clinical neurology, the psychologist must also know the individual neurologist or pediatrician with whom he is working. A characteristic which neurologists share with all other disciplines is that of idiosyncratic bias. This is merely to note that each individual neurologist or pediatrician has his peculiar strengths and weaknesses. There are areas within his specialty where he may nearly always be correct and other areas in which he may be quite prone to use his "waste basket" diagnostic category. An intimate knowledge of how the other fellow's mind works is surely as necessary in collaborative research as it is in other areas of human endeavor.

There is yet another and more formidable obstacle to productive research in the area of brain-behavior relationships that is uniquely psychological in nature. The problem may be tentatively identified as that of adding unique information to the already existing formulations of the neurologist, educator, or parent. It is hardly sufficient to identify a child as being brain injured or as having educational or adjustmental problems when these conclusions are apparent to even the most casual observer. The classroom teacher hardly needs to be told which of her children has educational problems, the parent does not need to be told that his child has difficulty in relating to authority figures, nor does the neurologist who has just finished examining a hemiparetic child need to be told that the youngster is brain injured. A psychologist's evaluation of an individual subject, or his report of a formally controlled and executed research investigation which is little more than a wordy restatement of findings obvious to everyone adds little either to the stature of our profession or to our fund of knowledge. The psychologist's job is to specify on the basis of independent findings the interrelationships between organic and behavioral deficits, to chart the probable course of development under certain specified conditions, and if possible to indicate remediational procedures designed to alter the course of development when such alteration appears desirable. Although this is admittedly a counsel of perfection, an examination of the requirements of this task may help to bring perfection and reality a little closer together. If it can be agreed that public as opposed to private knowledge ought to be the goal we seek, then one of the first requirements of the problem would seem to be a set of objectively scorable and interpretable tests bearing a known relationship both to neurologic and to behavioral deficits. In view of the complexity of the brain damage problem, both from the point of view of the neurological dimensions of dysfunction and from the point of view of the multi-dimensional nature of behavioral deficit, the tests the psychologist employs should presumably be as comprehensive as possible with respect to the functions they purportedly measure.

A battery of tests which has been shown to meet some but not all of the requirements listed above has been in use for several years at the Neuropsychology Laboratory of the Indiana University Medical Center. The remainder of this paper will describe this battery of tests, the validational evidence currently available, and some of the ways in which the battery of tests might be employed in various clinical settings.

The battery of tests to be described has been used repeatedly in the neuropsychology laboratory since 1953 for assessing the influence of brain lesions on intellectual and adaptive abilities in school age children. Some of the measurements (e.g., Wechsler-Bellevue, Halstead Finger Oscillation Test, Halstead Time Sense Test, Seashore Rhythm Test) have been repeatedly used in assessing cerebral lesions in adults and in that context have established validity (Reitan, 1955). The remaining measures consist of modified versions of the Trail Making Test, modificatons of three of the test procedures of the Halstead Battery of Neuropsychological Tests, the Dynamometer Test, and the Wide Range Achievement Test. The selection of tests for use with a school age population was dictated largely by the success with which the same or highly similar tests had been used in investigating cerebral dysfunction in adults. The modified tests were developed by Reitan in 1953 and in each instance consisted of revising the difficulty level of the original test to make it more suitable for use with a younger population. A brief description of the way in which each test was modified is given below.

The Category Test is a concept formation test requiring the subject to abstract and apply principles from serially presented visual stimuli. As developed by Halstead, the test consisted of seven subtests, each with its own organizing principle, utilizing a total of 208 visual presentations. The test was shortened to include only 168 presentations divided into six subtests. Although the original stimulus items were not used in their entirety, the same organizing principles or concepts were employed.

The Halstead Speech Sounds Perception Test consists of 60 spoken speech sounds presented to the subject via a tape recorder. The subject is required to match the sound he hears with one of four visually presented choices. The test is modified for use with younger subjects by deleting one of the possible alternatives, thus simplifying the discrimination required of the subject.

The Halstead Tactual Performance Test utilizes the Seguin-Goddard form board, requiring the subject to place the ten blocks in the correct spaces while blindfolded. The subject is first required to perform the task using only his dominant hand. A second trial, unanticipated by the subject, is then administered with the subject using only the nondominant hand. Finally, a third trial is given with the subject using both hands. Upon completion of the third trial, the form board and blocks are put away and the blindfold removed. The subject is then asked to draw an outline of the board, locating as many blocks as he can remember in their correct positions. The test yields three scores, one score for total time consumed during the three trials, a second score corresponding to the number of blocks correctly drawn from memory, and a third score for the number of blocks correctly localized in the subject's drawing. The modification of this test for use with a younger population consisted of eliminating four of the blocks, thus making it a six block test rather than the original ten block test.

The Trail Making Test is divided into two parts. The first part consists of 25 numbered circles and the subject is required to connect them in sequence as rapidly as possible. The second part of the Trail Making Test consists also of 25 circles but utilizes both an arithmetic and an alphabetical sequence. The subject is required to alternate between the two sequences (i.e., 1-A-2-B-3-C, etc.). Each part of the test is scored in terms of seconds required for completion. The test was modified by deleting ten of the circles in each case, thus reducing the total number of circles from 25 to 15 for each part of the test.

The Dynamometer Test uses a standard hand dynamometer. The strength of grip for each hand is recorded separately.

The Aphasia Screening Test is a modification of the Halstead-Wepman Test for Aphasia. The test is designed to survey a large number of possible aphasic deficits (e.g., dysnomia, dyslexia, dysgraphia, visual letter dysgnosia, etc.). Evaluation of results of this test requires considerable experience in its use with a wide variety of aphasic subjects.

A series of sensory perceptual examinations is also employed which is designed to elicit from the subject possible evidence of sensory suppression or imperception under conditions of simultaneous bilateral stimulation. In general, the procedure for these examinations is to stimulate first one side of the body and then the other with sufficient intensity to evoke a reliable response from the subject. When the minimal level of intensity of stimulation necessary to evoke a reliable response from the subject is determined, both sides of the body are stimulated simultaneously using first tactile, then auditory, and finally visual stimuli. Additional sensory examinations include a test for finger agnosia and a test for fingertip number writing perception. In the finger agnosia test, the patient is required to identify solely on the basis of tactile stimulation which finger of which hand is being stimulated. In the fingertip number writing perception test, numbers are written on the tips of the fingers, and the patient is required to identify solely on the basis of tactile information which number is being written. These sensory examinations are employed by many neurologists as a standard part of the physical neurological examination. Differences in the employment of these tests relate primarily to the degree to which administration and scoring are standardized.

The final series of tests employed in the neuropsychology laboratory requires the subject to reproduce simple geometric figures using a visual model as his guide. Although the subject's drawings are not formally scored, it is nevertheless possible to determine qualitatively the degree of constructional dyspraxia that may be present.

In addition to the above tests, an individually administered test of general intelligence is also routinely given to each subject. For adults, the instrument of choice is the Wechsler-Bellevue (Form I), whereas for children, the Wechsler Intelligence Scale for Children is used.

Three formally conducted studies focusing on the validity of all or part of the above battery of tests for assessing brain dysfunction in school age children have been completed. In the first study (Reed, Reitan, and Kløve, 1965), 50 brain damaged children ranging in age from 10 to 14 years were matched in pairs with 50 normally functioning children on the variable of age. The sensitivity of each test to the brain damage factor was determined by computing the appropriate *t* ratio and by determining the proportion of cases in which the brain damaged children performed less adequately than their matched controls. The brain damaged children performed significantly less well than the control children on all of the tests, with difference between the two groups occurring more frequently on tests of language functions than on other of the testing procedures.

A second study (Reed and Fitzhugh, 1966) employed four groups of brain damaged subjects (mildly impaired children, moderately impaired children, mildly impaired adults, and moderately impaired adults) and appropriate control groups. The purpose of the study was to demonstrate under what conditions brain damaged children demonstrated patterns of intellectual and adaptive deficits similar to those obtained with brain damaged adults. The results of the study indicated that age of onset and chronicity of brain dysfunction were important determinants of the pattern of deficits demonstrated by the subject. Children with longstanding chronic cerebral dysfunction either of mild or moderate degree demonstrated patterns of deficits similar to those demonstrated by adults with chronic long term cerebral dysfunction. These three groups differed substantially, however, from a group of adults who had sustained their brain lesions during the years of adulthood.

A third study (Reed, 1963) compared 40 children known to have sustained brain lesions with 40 children suspected of having sustained brain lesions with 40 normally functioning children. The groups were matched in triads on the basis of chronological age. The group of children suspected of having brain lesions was referred to the medical center for evaluation of learning disorders. This group of children proved to be just as severely impaired on measures of language ability as the group of children known to have brain lesions, but fell at a point between the brain damaged and control children on many of the nonlanguage ability measures.

In all of the above studies, the diagnosis of brain damage was based on the results of the physical neurological examination, electroencephalographic tracings, and specialized diagnostic procedures in those cases where such procedures were indicated. It was not possible to compose groups of brain injured children that were homogeneous with respect to any single diagnostic category and thus the studies did not do justice to the rich variety of impairing neurological conditions represented in the groups. The studies did demonstrate, however, that the effects of the brain lesions were routinely apparent on the psychological tests administered to the subjects and on that basis a strong argument can be advanced for the usefulness for such a battery of tests in studying groups of neurologically impaired children

An additional feature of using such a battery of tests which is not apparent in any of the formal research studies relates to the use of the tests for understanding the consequences of the brain lesions for the intellectual development of an individual child. Since the clinical utility of this particular test battery has never been formally demonstrated, any statement as to its usefulness in understanding the effects of brain lesions on the intellectual development of a particular subject must necessarily be in the form of a testimonial based on experience with many hundreds of cases. For each of the hundreds of children whom our technicians examine with this battery in the neuropsychology laboratory, a formal report is written in which the test results are used to infer the presence or absence of disordered brain functions. Assuming that evidence in the test results suggests that a brain lesion is present, every effort is then made to determine whether the lesion is diffuse or whether it is well localized in character, whether it is static or progressively disabling in course, and whether the lesion has exerted clinically significant effects on the intellectual and adaptive abilities of the individual. All reports are written on a blind basis, that is without knowledge of any other information concerning the patient apart from the patient's age, education and test results. We have been routinely successful in identifying the presence of brain lesions, whether they are diffuse or localized in character, whether they are acute or chronic in course, and the effects of the brain lesion on the current status of the patient's intellectual resources. We have also tried to give a very practical orientation to our evaluation of brain injured subjects, indicating for example whether or not the patient can be expected to maintain normal academic progress under conditions of routine classroom instruction, whether or not the development of serious behavior problems should be anticipated, etc. Inferences of this kind are possible partly because of the extensive experience we have had with this particular battery of tests and its applications, but partly also because of the variety of ways in which the data may be approached. The test data may be initially approached, for example, from the point of view of the typical level of performance of the subject. The validity of this approach is based on the fact that brain injured children routinely perform less adequately on this battery of tests than do normally functioning children. In selected instances, however, this approach may have little merit either because an occasional brain injured subject may do very well on all of the tests or because the deficits apparent in the general level of performance may be more reasonably attributed to factors other than brain injury. A second and more useful approach to the test data involves the comparison of test results obtained from the two sides of the body. The Finger Tapping Test, the form board test, and the various sensory examinations all permit such comparisons thus enabling the subject to act essentially as his own control. Many of the sensory-motor tasks that the subject is required to take are suffciently simple in terms of their mental age requirements that reliable results can be secured from quite young or quite seriously impaired subjects. Inadequate performances on these series of tests, particularly if the sensory motor deficits are reasonably well lateralized, can often be used as a secure anchoring point for inferring the concept of brain dysfunction or damage. With the initial inference of brain damage securely anchored, one can then proceed to trace the consequences of the brain lesion on tests of so called higher mental abilities. Unless one has a comprehensive battery of tests, however, it is very difficult either to anchor one's interpretations or to trace the effects of brain lesions in the variety of ways necessary for clinical application. The following case material illustrates the kinds of inferences that can be drawn from neuropsychological test data. The original interpretations of the test results have been modified slightly in an effort to make the interpretations easier to follow.

The subject is a girl, J.B., age approximately 10 years, who had completed three years of school. She was involved in an auto accident in which she sustained a left sided skull fracture about six months prior to the initial neuropsychological evaluation. Following the accident, J.B. was unconscious for more than six weeks. Her recovery was additionally complicated by two episodes of meningitis. The second neuropsychological evaluation was performed about eight months after the initial examination and illustrates the use of serial psychological testing in evaluating the recovery course of the patient.

This 9 year 11 month old patient scored in the borderline range of psychometric intelligence as noted above with performance abilities being slightly superior to verbal skills. On tests designed to be specifi-

TABLE 1

Report of Neuropsychological Examination Initial Testing

Wechsler Intelligence Scale for Children	*Score*	*Halstead's Tests*	*Score*
VIQ	74	Category Test	59
PIQ	80		
F-S IQ	75	Tactual Performance Test	
Verbal Weighted Score	29	Right Hand 10.0′ (2 in) Time	22.1*
Performance Weighted Score	36	Left Hand 7.0′ (all in) Memory	2
Total Weighted Score	65	Both Hands 5.1′ (all in) Location	1
Information	6[a]		
Comprehension	5[a]	Seashore Rhythm Test	10
Digit Span	8[a]	Raw Score	11
Arithmetic	6[a]		
Similarities	5[a]	Speech Perception Test	28
Vocabulary	5[a]		
Picture Arrangement	8[a]	Finger Tapping Test	
Picture Completion	8[a]	Right Hand	23
Block Design	9[a]	Left Hand	32
Object Assembly	4[a]		
Digit Symbol	7[a]	Time Sense Test	
Mazes	7[a]	Memory	354.7
		Visual	741.6

[a] raw scores

Trail Making Test

Trails A II	— 32″	(0 errors)
Trails B II	— 71″	(2 errors)
Trails Total	— 103″	(2 errors)

Aphasic and sensory perceptual symptoms: spelling dyspraxia, mild central dysarthria, and right-left confusion. The difficulty that the patient had in spelling certain words may conceivably reflect the failure in acquisition of certain language skills, but the types of errors that she made were sufficiently unusual that we are inclined to think that this represents a genuine dysphasic loss. For example, the patient spelled the word "square" as "crar" and the word "cross" as "choar." These are not the kinds of errors that a child just learning to spell typically makes. The patient also demonstrated certain perceptual disorders including a consistent tendency to suppress a tactile stimulus to the right hand when it was given together with one to the left side of the face. With the reverse combination (i.e., left hand and right face), the patient made no errors in reporting double stimulation. The examination for sensory imperception could not be performed with visual stimuli because of total loss of vision in the right eye. On both the finger agnosia examination and the fingertip number writing perception there were mild to moderate bilateral impairments. The patient also demonstrated mild perceptual impairment in her attempts to copy simple geometric figures.

cally sensitive to the organic condition of the brain, the patient's performances ranged in quality from ones which were at a near normal level of adequacy to performances which were moderately impaired. On the Category Test, for example, which is normally one of our more sensitive indicators of the organic condition of the brain, the patient's performance was only slightly below the expected level for her age. However, on the Tactual Performance Test, the Seashore Rhythm Test, the Speech Perception Test, and the Finger Tapping Test, the patient's performances were clearly in the range characteristic of patients with clinically established brain lesions. On all tests directly dependent on intactness of motor functions (Tactual Performance Test, Tapping Test, Dynamometer Test) the patient's performances were clearly impaired and in each case the right upper extremity was inadequate relative to the left.

The pattern of test results for this patient clearly indicated at least mild impairment of intellectual and adaptive abilities and there was substantial evidence that this impairment was directly attributable to cerebral damage. The right sided sensory and motor involvement clearly indicated that the left cerebral hemisphere was functioning on a much less adequate level than the right. However, there were mild indications for right hemisphere dysfunction as well (constructional dyspraxia, bilateral involvement on the finger agnosia and fingertip number writing perception tests), and thus the overall picture was one of an area of maximal involvement in the fronto-parietal region of the left cerebral hemisphere superimposed on mild diffuse cerebral dysfunction. Although we cannot specify with any certainty the type of neurologic impairment that is reflected by these test results, the pattern would be most consistent with traumatic injury to the brain maximally affecting the left cerebral hemisphere. The indications of rather focal involvement within the left cerebral hemisphere (the sensory imperception findings especially) suggested that the brain lesion is of relatively recent origin. But on the other hand, the patient's test performance on the Category Test suggested that there has been at least some recovery of basic adaptive abilities. The patient should be reexamined in this laboratory within six months of the data of the present testing.

The Wide Range Achievement Test was also administered to this patient, yielding a Reading Placement Grade of 3.4 years and a Spelling Placement Grade of 2.5 years. Both of these scores suggested the possibil-

TABLE 2

Report of Neuropsychological Examination—Testing II

Wechsler Intelligence Scale for Children	*Score*
VIQ	76
PIQ	72
F-S IQ	72
Verbal Weighted Score	31
Performance Weighted Score	30
Total — Weighted Score	61
Information	5
Comprehension	5
Digit Span	6
Arithmetic	5
Similarities	10
Vocabulary	6
Picture Arrangement	2
Picture Completion	7
Block Design	9
Object Assembly	6
Digit Symbol	4
Mazes	8

Halstead's Tests			*Score*
Category Test			66
Tactual Performance Test			
Right Hand	8.5	Time	15.1
Left Hand	4.7	Memory	4
Both Hands	1.9	Location	1
Seashore Rhythm Test			10
Raw Score			14
Speech Perception Test			21
Finger Tapping Test			
Right hand			25
Left Hand			32
Time Sense Test			
Memory			
Visual			

Trail Making Test

Trails A II	— 30″	(0 errors)
Trails B II	— 130″	(3 errors)
Trails Total	— 160″	(3 errors)

Asphasic and sensory perceptual symptoms for the 10 year, 7 month old female patient (Table 2) follow: spelling dyspraxia, central dysarthria, and right left disorientation. The examinations for sensory imperception yielded normal results with auditory and tactile stimuli. The examination could not be performed with visual stimuli because of loss of vision in the right eye. On the finger agnosia test, the patient was moderately impaired with the left hand, and mildly impaired with the right hand. On the fingertip number writing perception test, the patient performed at an essentially normal level with the left hand, but was clearly impaired with the right hand. A mild but definite constructional dyspraxia was apparent in the patient's attempts to copy simple geometric figures.

ity of mild educational retardation, but they represented a clearly higher level of achievement than was typified by the other test performances. This type of pattern is fairly typical for patients who have sustained relatively recent insults to the brain but who have a history of normal functioning up until the time of the brain injury. It is our opinion that this patient will have considerable difficulty in maintaining normal academic progress in the future under conditions of regular classroom instruction. It is quite possible of course that there will be additional recovery of mental abilities as the patient's clinical condition continues to improve, and at the time of our next examination we should be able to determine somewhat more precisely the patient's chances of maintaining reasonably normal educational progress.

This patient scored in the borderline range of psychometric intelligence earning a full scale IQ of 72 with verbal and performance IQ's of 76 and 72, respectively. Analysis of the subtest scores revealed a very poor performance on the picture arrangement subtest. On the tests of adaptive abilities, the patient's performances ranged in quality from borderline to moderately impaired. Clear motor involvement of the right upper extremity was apparent on the Tactual Performance Test, the Finger Tapping Test, and the Dynamometer Test. On none of the tests administered to her did the patient achieve a normal level of functioning.

The pattern of test results for this patient provided clear evidence of organic impairment of intellectual abilities. There was consistent evidence in the test results that the left cerebral hemisphere was functioning on a less adequate level than the right, but there was evidence of mild right hemisphere dysfunction as well. The pattern of results is typical for patients who have sustained severe head injuries but who have recovered from the acutely impairing effects of the trauma at the time of the testing.

This is the second full set of test results which we have obtained from this patient during an interval of slightly more than eight months. Comparison of the two sets of test results reveals that both the motor and sensory deficits involving the right side of the body are much less pronounced now than they were at the time of the earlier testing, but there has been no corresponding improvement in the patient's higher mental abilities. In other words, the test data suggest that the biological condition of the brain has stabilized somewhat during the intertest interval, but the anticipated gains in higher mental abilities have not occurred. The patient was also administered the Wide Range Achievement Test on both occasions, the values at the time of the first testing being 3.4 years for the Reading Placement Grade and 2.5 years for the Spelling Placement Grade. There has been no change in those values during the intertest interval.

We have independent knowledge that this patient sustained a severe head injury as a result of an automobile accident in July of 1962. There can be little doubt that the organic impairment of intellectual abilities which is so apparent in both sets of test results is directly attributable to the head injury. Although it is not possible to state in any exact terms the degree to which this patient has been impaired, some indication of the degree of impairment can be gained from the Wide Range Achievement Test. The scores which this patient earned on this test at the time of the first testing clearly suggest that the patient had derived essentially normal benefits from her formal educational experiences. It is quite clear from the test results that this patient cannot be expected to achieve at anything approaching a normal level. If the patient is returned to a regular public school environment, she will require extensive individualized help and tutoring if she is to maintain even minimally satisfactory progress. The patient's current IQ values are in the range normally considered as appropriate for special education classes, and these classes would very probably provide a more appropriate environment for the patient than would a regular public school classroom. This recommendation, of course, is based on the patient's current level of functioning. Although it is possible that the patient will demonstrate some progress in terms of recovering higher mental abilities, it should be noted that more than a year has now elapsed since the time of the accident and the probability of any sudden change for the better in terms of this patient's general level of functioning is quite remote. We would like very much to reexamine this patient within one year of the date of the present testing or sooner than that should there be any significant change in her clinical condition.

Relating Neuropsychological Data

Formal research studies already completed have demonstrated the potential usefulness of the battery of tests for establishing firm relationships between neurological inferences and psychological test performances. Extensive clinical experience has provided personally convincing evidence of the usefulness of the test battery in understanding the constellation of problems unique to the individual subject. It yet remains to be seen, however, whether or not the same test data can be used in relating effectively to the social and educational problems which many children with brain lesions experience.

The route to success in relating neuropsychological test data to the social and educational problems which brain injured children experience requires first of all an adequate classification scheme for the dependent variables. Since the behavior of the child in the classroom is a reasonably specifiable segment of behavior that reflects both educational and interpersonal problems, it is to this ubiquitous life experience that psychologists might most profitably direct their attentions. Fortunately for progress, this kind of environment is far less alien to the psychologist than is the world of the clinical neurologist described earlier. Considering first the class of variables known collectively as "educational potential" or "educational achievement," most psychologists have at least passing familiarity with the standardized, objective, group administered tests routinely employed by teachers in assessing the educational development of children. These kinds of measures, together with the rating scales and check lists frequently employed by many teachers, offer a very natural starting point for the development of a clinical research program relating neuropsychological test data to classroom behavior. The basic problem to which such a program would address itself is the nature and extent of the relationships between neuropsychological test data and educational achievement. By inference, of course, such a program would also relate the behavioral deficits displayed in the classroom to neurological deficits. Such a research program could be an invaluable extension of our general knowledge concerning intellectual functions by providing three anchor points to which such functions could be tied: neurologic impairment, psychological test results, and educational progress.

In addition to these three principal anchoring points, two additional kinds of measurements could be developed within the classroom environment. The traditional kinds of sociograms depicting certain aspects of the nature and extent of a child's interpersonal relationships could be a valuable opening wedge into the more general problem of relating ability measurements to adaptive or maladaptive behavior in peer relationships.

A second class of measurements that could be developed within the educational setting relates to the ways in which educational and social competence, or the lack thereof, can be modified. The standard armamentarium of classroom teachers includes extensive training in the techniques of education. These techniques are presumably ways in which the teacher modifies the behavior of her pupils, but there is little direct evidence concerning the efficacy of such techniques. It should be noted at this point that there is no necessary relationship between the etiology, nature, and extent of a child's behavioral deficits and a rehabilitational program designed to ameliorate these deficits. It is both reasonable and heuristic to believe, however, that logical relationships do indeed exist between diagnosis and remediation. A neuropsychological educational clinical research program could conceivably be an admirable vehicle in which systematically to explore such beliefs.

In conclusion, the hope of relating neuropsychological test data to educational and social problems assumes that the abilities a child has are important determinants of his educational achievements and also of the extent and quality of his interpersonal relationships. Additionally, such a hope rests on the belief that educational and social behavior can be meaningfully dimensionalized. In other words, an elaborate classification system of human abilities, even if firmly anchored to an equally elaborate neurological description of brain function and dysfunction, will be of very limited use unless both classes of variables can be effectively related to empirically sound and socially meaningful dimensions of behavior. It may be argued with impeccable logic that intellectual abilities are important determinants of the ways in which children solve problems and of the adequacy with which they solve problems regardless of whether these problems are educational or interpersonal in nature. But it is with science as it is with other human schemes: "the proof of the pudding is in the eating" and until the great distance between impeccable logic and demonstrable empirical relationships is traversed, the value of even the most promising neuropsychological evaluations of children will remain largely untested.

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Screening Children with Cerebral Dysfunctions through the Laboratory Method

LUCIANO L'ABATE

The major components of the laboratory method in evaluation are: (a) standard test batteries; (b) specially trained and supervised subprofessional assistants to administer the tests; and (c) a clinical psychologist to supervise the assistants. The division of labor implies a differentiation of technical and professional skills and responsibilities, separating test administration and scoring from interpretations of test results, report writing, and consulting with referring sources, agencies, and parents. This method has the advantage of considerably lowering psychodiagnostic evaluation costs from an estimated $150.00 to about $50.00, increasing the efficiency of the clinical psychologist manyfold, and allowing him to evaluate as many as six to eight children a week on a part time basis. An added advantage of this method is that the collection of information according to standard operating procedures links, rather than separates service and research functions (L'Abate, in preparation, 1964a, 1964b).

The methodological viewpoint basic to the laboratory method is called multiple operationism. This viewpoint assumes that it takes more than one test, technique, or treatment to understand and to change behavior. By this token, each major psychological function is evaluated by at least two or more tests, depending on the amount of time available. Four hours are estimated as the optimal time needed to evaluate a child, excluding, of course, both breaks and interruptions between sessions which are one hour or less in length.

Since the screening of most children occurs in settings where a variety of questions are asked, the clinical psychologist must be ready to evaluate four major aspects of functioning: (a) verbal and symbolic functioning, (b) visual motor performance, (c) learning deficits and academic achievement, and (d) emotional, psychosexual, and interpersonal adjustment. The interrelatedness of these four aspects makes it imperative to view cerebral dysfunctions in children as affecting all of these functional areas. Consequently, cerebral dysfunctions cannot be studied in and by themselves. Of course, greater emphasis on various parameters of localization and specificity would require the kind of battery and neurological study reported by Reed elsewhere in this special publication. His major interest lies in the specification of the nature of the damage in a neurosurgical setting where clear cut external criteria on each child are carefully collected. Our major problem, instead, is to screen for cerebral dysfunctions in educational, pediatric, and psychiatric settings. Thus, at best, our diagnostic conclusions can only cover the presence or absence of the cerebral dysfunction, with some general inferences concerning its severity, lateralization of functions, and overall prognosis.

Working at a distance from the child, as in the laboratory evaluation, requires that safeguards in the form of complete batteries be administered routinely, unless the child is impossible to evaluate or he does not complete the evaluation. Furthermore, assistants are required to keep a running set of notes concerning the child's attitudes and extra test responses to each test, within each session and between sessions, as will be shown in the two case studies presented here. Besides the requirement of at least two tests for each of the four aspects of functioning, we have devised standard test batteries for three age levels: (a) 3 years to 5 years and 11 months, (b) 6 years to 8 years and 11 months, and (c) 9 years to 14 years and 11 months.

To study the four aspects of psychological functioning, we employ old and new tests, which may be already familiar to the reader. We shall review some of the findings of those techniques that in the psychodiagnostic battery are particularly useful in the screening of cerebral dysfunctions in children.

The Analytical Approach: A Few Selected Tests

The Frostig Developmental Test of Visual Perception. This is a relative newcomer (Frostig et al., 1964; Maslow and Frostig, 1964; Maslow, et al., 1964) to the overall battery for the evaluation of brain damage. Its newness and good standardization do present various advantages which are not present in more impressionistic types of tests, such as the Bender-Gestalt. Although it may take a longer time to administer, its usefulness as a clinical as well as a research tool is established—with a proviso. As Corah and Powell indicated (1963), two general factors of intelligence and age would account for most of the variance. This finding suggests that, while scatter analysis of the various subtests may be of dubious value, its perceptual quotient as an overall measure is a useful diagnostic index of cerebral dysfunction and educational potential. Space does not allow greater detail concerning the nature of this test, but it is strongly recommended as a welcome addition to our armamentarium.

The Illinois Test of Psycholinguistic Abilities. This test was originally developed by James J. McCarthy and Samuel A. Kirk. It has been used with mental retardates by Gallagher (1957). Bateman (1964) reviewed most of the research done with this excellent addition to the test battery. In a factor analysis of the ITPA with other miscellaneous and primary mental abilities, Center (1963) found that the ITPA represented itself in seven of eight factors derived from a variety of variables. As Kass (1962) suggested, this test is not only excellent for the differential diagnosis of learning deficits, but within the overall battery is an excellent tool to screen for various types of brain damage. However, it should be emphasized that the use of this test without other tests, like the Frostig Test of Perceptual Development, would limit the evaluation of children to a very narrow band. Consequently, although the ITPA is another welcome addition to available tests, it should be used in a systematically devised battery.

The Minnesota Percepto-Diagnostic Test. This is a short derivation of the Bender-Gestalt which would seem to be a more objective development than the Bender-Gestalt, although it yields a single score on the basis of the rotation of figures (two figures with three different backgrounds). Its usefulness as a screening test for brain damage needs to be assessed more carefully (Fuller and Shaw, 1963; Fuller, 1964, 1965).

Draw-A-Person Test. Another part of this battery for children below six years of age is the DAP scored according to Goodenough's system. As soon as Harris' (1964) revision and extension of the Goodenough system becomes more familiar, it will be possible to use existing data for an evaluation of its usefulness in assessing brain damage. As Clawson (1962) suggested, a battery composed of the WISC, the Wide Range Achievement Test, the Draw-A-Person Test, a sorting test of her own device, the Bender-Gestalt, and the Benton Right-Left Discrimination Test would furnish sufficient bases to screen children of average intelligence (IQ scores between 91 and 123) for brain damage. Her conclusion was based on an initial battery of 22 tests.

Hidden Figures Test. Another test used for the discrimination of brain damage is a modification of Gottschaldt's Hidden Figures Test (Goodenough and Eagle, 1963; Teuber and Rudel, 1961, 1962). This researcher's modification, however, pertains to changing the stimuli and responses from right to left to screen for possible lateralization of functions. Atlhough this researcher has used this test extensively, he feels that the time involved to administer it, approximately twenty to thirty minutes, does not warrant its use as a routine instrument. He is planning to demote it to supplementary status and to use the Kahn Test of Symbol Arrangement (KTSA) as part of his evaluation of children between 9 and 15 years of age. For the time involved, the KTSA would appear to give much more information (L'Abate and Craddick, 1965).

Wechsler Intelligence Scale for Children. Since the review of Littell (1960), which brought into question this test's validity in evaluating cerebral dysfunctions in children, a variety of studies have come up, but the results still remain indefinite (Rowley, 1961). Wilke (1963), for instance, studied 25 brain damaged children between 6 and 12 years of age with normal verbal IQ scores. They were given the Arthur Point Scale as an index of visual-motor efficiency. From this type of evaluations he hoped to predict school readiness, but results were not given in the abstract.

Hopkins (1964) made an analysis of the WISC scatter patterns of what he called "carefully identified children with normal IQ and neurological impairment." Although he found several significant differences among mean scores of subtests, these differences were found to have limited diagnostic significance for the individual case, due to the low reliabilities of these scores. Hopkins explained his negative results in terms of the expected homogeneity for profiles as heterogeneous as those resulting from brain damage. This point is well taken and should perhaps be kept in mind in any clinical evaluation of children, especially when using the WISC. Although impressionistic studies of the type of Clements and Peters (1962) would indicate various patterns for minimal brain damage, Hopkins' results uphold a more critical examination of the WISC, as in the work of Caputo, Edmonston, L'Abate, and Rondberg (1963) and Rowley (1961).

Burns' results (1960) using retarded children are perhaps a little more satisfactory. He matched two groups of children selected from 1,400 consecutive cases in a children's hospital. One group consisted of children who were normal until the age of three but had subsequent central nervous system involvement and tissue injury as substantiated by neurological data. The second group consisted of children with known brain tissue underdevelopment and maldevelopment. Burns found consistent differences in the WISC subtest scores, both between the two groups and individually. The tissue injury group was characterized by a pattern of block design score below the object assembly score. The tissue underdevelopment and maldevelopment group was characterized by a block design score above the object assembly score, with lower similarities, arithmetic, and picture completion scores and also a suggestion of slower EEG frequencies, indicating a greater immaturity of the brain. As other investigators have suggested, concomitant biochemical, neurological, and psychological approaches may improve our diagnostic, prognostic, and possibly even our etiological knowledge of brain dysfunctions.

Caputo, et al. (1963), studied samples of brain damaged children with seizures and children with nonseizure diffuse brain damage, matching them individually with controls drawn from the same files. The results showed that the socioeconomic level of the children was a major factor influencing their functioning. The picture completion subtest was the only significant subtest pattern to differentiate the seizure from the nonseizure brain damaged children. Although block design and digit symbol subtests were sensitive to brain damage, they were influenced by psychopathology as well.

Wunderlin and McPherson (1962) used the WISC in a different but still important approach. In studying the sensitivity to imbalance of normal and anoxic brain damaged children, they noticed that the brain damaged child's approach to the WISC was marked by comments about the precariousness of the objects and the higher incidence of inactivity. Anoxic subjects manifested a sensitivity to imbalance, which was shown through soft test attitudes, such as the forecast of lack of integration evidenced in the house of the picture arrangement subtest, or the interchanging of the manikin's legs in the object assembly subtest. This kind of observation would be of greater significance to the clinician than to the researcher but should be considered nonetheless.

Solkoff (1964) is responsible for one of the few experimental studies on the effects of frustration on coding performance. He applied three degrees of frustration to 36 brain damaged boys. High frustration consisted of interruption of the task and subsequent withholding of a promised reward. This type of frustration produced a great deal of impairment in the performance of brain damaged children and an increase in errors. This type of study, using clinical tools for the experimental manipulation of each of the subscales, should be encouraged.

Figure Reproduction Tests. The Bender Visual Motor Gestalt Test is not used in the Atlanta laboratories on the basis of its unclear scoring criteria and of several suggestions in the literature that some other tests can be just as useful. We feel that the Frostig Developmental Test of Visual Perception is a definite improvement over the Bender-Gestalt which forces one to arrive at an impressionistic analysis. Billingslea (1963) concluded that there is still a great deal of work to be done with the Bender-Gestalt before we can accept its validity.

Keogh (1963) found that the total score was the only appropriate basis for evaluation, but she reported the Bender-Gestalt to be of limited value as a diagnostic test for reading difficulty, even though she felt that it might be a potentially useful test for the identification of potentially good readers! Koppitz (1962) studied 103 children from 5 to 10 years old. She found that the total score differed among age groups, suggesting that the diagnostic usefulness of the Bender-Gestalt varies a great deal with age. Smith and Keogh (1962) checked on this particular hypothesis by testing 117 public school children from kindergarten to second and third grades. Bender-Gestalt protocols were scored according to two objective scoring systems together with a clinical checklist. Although no sex differences were found at any grade level, there was an improvement among grades for both scoring systems and six of eight clinical checklist items. The greatest change in performance occurred between kindergarten and second grade, with only a minor improvement between second and third grades. As they suggested, the Bender-Gestalt could be used as a group measure of developmental differences in the first grade. Its clinical usefulness on an individual basis is questionable when we have better instruments available.

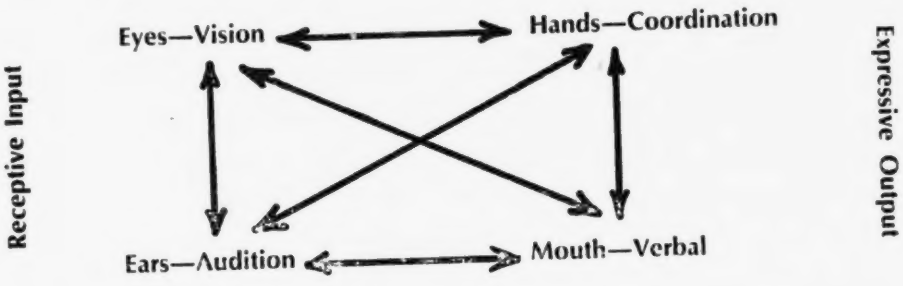
Instead of the Bender-Gestalt, we use Benton's Revised Visual Retention Test (Form C) as it has a much more objective scoring system and is much more likely to reveal lateralized damage (Rowley and Baer, 1961). We also use Benton's right left discrimination for children between 6 and 9 years old (Benton, 1959).

Miscellaneous Tests. A variety of other tests seem to show promise in screening for brain damage in children. Some of these are rather ingenious, such as Rich's (1963) tactual version of Raven's Progressive Matrices which he used for the study of blind children. Others are: Archimedes Spiral and the Trail Making tests (Davids, Goldenberg, and Laufer, 1957), Tien and Williams' (1965) Organic Integrity Test, and the Kahn Test of Symbol Arrangement (L'Abate and Craddick, 1965). Graham and Berman reviewed a variety of techniques (1961) for testing brain damage in preschool children. They summarized that it is only in the last decade that interest has extended to children in the preschool age range. It is probable that the next decade will see even more marked progress. As they suggested, "Measurement difficulties lie less in the ability of investigators to devise ingenious techniques than in stubborn problems of defining a brain injured group." They suggested that longitudinal studies may help to clarify some of the controversial questions of definitions and may be "particularly valuable in determining whether or not undetected brain injury at birth contributes to a variety of later defects, including the frequently described clinical syndrome of hyperactivity, distractibility and impulsivity."

The use of group screening techniques for the identification of brain damage should be explored, as in a work of White and Phillips (1964). They had children copy figures from the blackboard to screen for gross and fine coordination. Using these methods during four years of study from prekindergarten to the sixth grade and testing predictions against later academic achievement, both authors felt that these kinds of techniques were highly reliable for predictions in the primary grade. L'Abate (in preparation), used a method of lantern presentation for the Benton Revised Visual Retention Test (Form E) and figures from Seguin's Formboard, comparing the relationship of performance on these tests to Raven's Progressive Matrices and the Stanford-Binet Vocabulary IQ's. Finally, comparative data collection at all levels and in all disciplines, but especially in clinical psychology, should lend itself to computerized data processing that will allow a quicker and more complete analysis of all factors involved, as suggested by the work of Eiduson, Johnson, and Rottenberg (1965).

The Global Approach

An Introduction to Interpretations. Before entering into specific case studies, a brief introduction to an interpretative approach of battery based test results is necessary (L'Abate, in preparation). This approach is summarized in Figure 1 in terms of major input channels of reception and output channels of expressions. The relationship between these two major aspects of functioning is then summarized in the outline underneath the figure. Normality implies superiority of reception over expression; we receive more than we can express. Dysfunction is presumed whenever the relationship between reception and expression is disturbed as in the four deductions outlined under Section B. Besides clear cut retardation or borderline intellectual functioning, two major classes of intervening variables (under C) may disturb the relationship between reception and expression: emotional disturbances and cerebral dysfunctions. For the purposes of this presentation, the concentration will be in demonstrating the lawful variability of indices and consistency of patterns in the case of cerebral dysfunctions.



A. Normality: Reception > Expression
B. Dysfunctions
  1. Reception ≠ Expression
  2. Vision > Audition = Coordination > Verbal
  3. Vision < Audition = Coordination < Verbal
  4. Vision = Audition ≷ Output (Verbal ≠ Coordination)
C. Intervening Variables
  1. Emotional Problems
    a. Random variability in indices
    b. Inconsistent patterns
  2. Cerebral Dysfunctions
    a. Lawful variability of indices
    b. Consistency of patterns

FIGURE 1. *The Laboratory Bases of Psychological Evaluation: A Simple Model for Battery Construction and Interpretation*

One example of how some degree of lawfulness may emerge from a variety of indices is illustrated in Table 1. This table illustrates further the usefulness of a wide band battery of tests. Although these eight children were referred from a clinic devoted to the study of hyperkinetic behavior, they differed considerably in their overall psychological functioning. This table illustrates also the usefulness of the reception expression distinction in its relationship to test behavior and EEG functioning. It is assumed that the Full Range Picture Vocabulary Test (which we have now replaced with the Peabody Picture Vocabulary Test (PPVT) is one of the many indicators of receptive functions, other indicators being the auditory visual decoding subscales of the ITPA. As suggested by the model given in Figure 1, ratios between receptive and expressive functions indicate various diagnostic consequences for the interpretation of battery results gathered through the laboratory method.

TABLE 1

An Exploratory Study of Hyperkinetic Behavior in Boys

			IQ's			*Benton*		
Subjects	*Age*	*FRPVT*	*Stanford Binet Vocabulary*	*Frostig Perceptual Quotient*	*H-F*	*C*	*E*	*EEG*
M.B.	5-2	105	100	0	0	0	>15	N.F.[a] (+)
W.R.	11-0	133	135+	100+	54	6	8	N.F.[b]
R.C.	7-8	75	114	85	8	2	15	L-Temporal
J.F.	8-1	70	100	56	0	0	>15	R-Occipital
E.T.	6-8	23 (KIT 121)	133	79	10	1	18	Non Specific (+)
B.B.	10-5	115	80	98	38	5	7	N.F. (−)
D.B.	6-6	101 (VA 83)	31[b]	68	0	0	21	N.F. (−)
C.W.	14-0	82	70	86	34	2	13	N.F. (−)

[a] Nonfocal
[b] Questionable

In the first two children the ratios of verbal input to output (FRPVT-Stanford-Binet Vocabulary) were approximately equal. Nonetheless, the first child could not perform (because of his young age) on the Frostig, Hidden Figures, and the Benton. This was not a fair comparison for his age. His EEG was nonfocal but abnormal. The EEG of the second child (W. R.) was nonfocal but questionable. The next three children (R. C., J. F., and E. T.) showed a strong discrepancy between low vocabulary reception and high vocabulary expression. Each of them received a Frostig score which was indicative of cerebral dysfunction as were their scores in Hidden Figures and Benton performance; their scores were much below what one would expect for the first two children. Two of the three children (R. C. and J. F.) showed focal EEG abnormalities. The third child's (E. T.) EEG was nonspecific.

The last three children (B.B., D.B., and C.W.) showed a pattern of high receptive verbal functioning (D.B.'s Van Alstyne IQ was 83) and low expressive functions. Their scores on tests of brain damage were strongly influenced by age, but their EEG's were all negative and nonfocal. This group illustrates how seemingly homogeneous children can be distinguished according to various patterns suggested by the model given in Figure 1.

The Practical Application—Individual Case Studies. Cases were selected to illustrate the usefulness of the laboratory method in using a wide band test battery. These results were gathered by Miss Jacqueline Azar, under the author's direction, instruction, and supervision. Testing and scoring were performed without her knowledge of the reason for referral and of the background information and history of the child.

Case Study 1

M.M. was referred by the chief of a pediatric hospital because of her inability to learn to read and to do arithmetic. The mother reported that the child was retarded; she based this conclusion on three IQ tests administered by school psychologists in her hometown. However, all concerned felt that a more specific degree of evaluation was needed. The mother was unaware of the child's retardation until the first grade. She was considered the healthiest of three daughters. In the spring of 1963, a schoolteacher asked the mother to have a pediatrician evaluate the girl, but he felt that there was "nothing wrong with her." However, the girl was not promoted to the next grade. In the fall of the same year the teacher worked extra time with her, and thyroid treatment was started by another pediatrician. She was tested by a school psychologist who felt that she was immature, but no advice and no intellectual evaluation was given, according to the mother. The second time she was tested by a school psychologist who reported the retardation, but according to the mother, "Nobody told me about [M.M.] being retarded." Her school progress had been extremely slow because apparently she could not remember what she had read, although she remembered things she had heard. She did not retain arithmetic, but anything she saw she retained in detail. Another pediatrician recently intimated the possibility of mental deficiency, but no formal diagnosis was apparently ever made in this regard. The principal of the school suggested a school for educable mental defectives. This suggestion upset the mother a great deal, since she did not feel her daughter was that retarded.

M.M. was described as being very determined and obstinate, but she was not a behavior problem, although she had her share of fighting with the oldest sister. She had a slight concussion in the spring of 1963, with loss of consciousness for a few hours, while playing with other children. She hit the back of her head and her vision "went off" for a few hours. She came out of it within a few hours, but she continued to complain of slight headaches once in a while.

TABLE 2

Case Study 1a Summary Sheet—Observations of Child

Name: M.M. Sex: F Age: 8-3

Describe child (physically): Height of child approximately that of 6 year old, while bone structure seems even younger. Weight is approximately minimal to height. Energy level appears adequate, but activity level is moderately passive. Holds pencil properly, and there seems to be no deficiency in vision or hearing. Her speech is good and there is no problem understanding her except when her answer is not related to subject.

Describe his behavior: M. in general worked very quickly, was not talkative during tests, and was quiet and cooperative. When she did not know the answer to a problem, she simply did not respond regardless of repetition of question or rephrasing. Found it very difficult to say, "d.k." [don't know].

Note changes in behavior within each hour: Attention span held an exceptionally long time (almost 2 hours until break) and child continued to respond except in cases where she d.k.—when she would rock in her chair, twist her hospital name bracelet, or twist her hair. Anxious a good deal of time when threatened.

Note changes in behavior between hours: When M. was tired of a test or reluctant to proceed she terminated her responses though not obviously. Overanxious to proceed on some tests. Idea seems to be to get this over with as soon as possible. Sometimes seems unconcerned about performance (MCPS). During testing M. somewhat flat affect.

How did the child relate to you during testing? Realized testing had to do with her school placement. Resents fact that will have to return to 1st grade in public school. Anxiety was not so much in encountering stranger as in the test materials she could not handle. Talked easily about friends, family, and pets, etc.

Note any unusual characteristic of the child's behavior: Some signs of possible practice on Performance section of WISC. At least Subject's responses and remarks seemed to indicate it. Can spell name except on Performance part of WISC. Disturbed when I would not show her how it should fit together. Recognized past mistakes.

TABLE 3

Case Study 1a Summary Sheet—Psychological Evaluation for Use with Children: 6 years 0 months to 8 years 11 months

Name: M.M. Sex: F Age: 8-3

		MA	*IQ*
1. Draw-A-Person (DAP)	Boy	6-0	70
	Girl	6-0	70
2. Peabody (PPVT)		6-3	85

3. R—L Discrimination
No. Correct/Total=9½/32
R: 16 L: 8

4. Frostig

	Age Equivalent	*Scaled Score*
Eye Motor Coordination	5-9	7
Figure Ground	6-0	7
Form Constancy	4-9	6
Position in Space	4-6	5
Spatial Relations	7-6	9
Total	PQ	68

5. ITPA

	Language Age	*Scaled Score*
Auditory Decoding	8-10	.42
Visual Decoding	7-3	−.78
Auditory-Vocal Association	6-10	−1.25
Visual-Motor Association	8-3	−.11
Vocal Encoding	4-5	−2.05
Motor Encoding	6-10	−.71
Auditory-Vocal Automatic	6-10	−1.65
Auditory-Vocal Sequence	4-2	−2.74
Visual-Motor Sequence	4-10	−2.25
Total	6-2	−2.73

6. Wide Range Achievement

	Grade	*EA*	*EQ*
Reading	1.2	6-0	70
Spelling	1.0	5-9	67
Arithmetic	1.4	6-3	73

TABLE 3—*Continued*

7. WISC

Information	5	V. IQ 75
Comprehension	8	P. IQ 92
Arithmetic	6	Full IQ 81
Similarities	6	
Vocabulary	8	
Vocabulary IQ	87	
Digit Span	3	
Coding	10	Ratios
Picture Completion	5	1.———
Picture Arrangement	12	2.———
Block Design	8	3.———
Object Assembly	9	
Mazes	—	

8. MCPS

	Raw	*Norms*
Conformism	16	22
M — F	12	50
Maturity	18	66
Aggression	6	22
Withdrawal	12	33
Hypermotility	7	58

9. Rorschach ———————————

Supplementary Tests

10. Family Relations ———————————

	Time	Cred.
11. Trail Making A	———————	———————
B	———————	———————

12. CAP

	MA	*IQ*
13. Stanford-Binet	———————	———————
S-B Vocabulary	———————	———————

M.M.'s test behavior and test results showed a strong discrepancy in her intellectual functioning. Although her visual motor problem solving performance, based on eye hand coordination, was considered to be within the borderline normal range (WISC Performance IQ = 92), her verbal expressive functions were relatively retarded in the dull borderline defective category (WISC Verbal IQ = 75). Her overall intellectual functioning, because of this discrepancy, was considered to lie within the dull normal range (Peabody IQ = 85; WISC Full Scale IQ = 81).

Even within the area of visual motor functioning, one should qualify the conclusion of borderline normality to indicate that even some aspects of her visual motor functioning were defective, especially when emphasis was given only to the visual input aspect of the visual motor functioning. M.M. functioned best when she was required to coordinate her eyes with her hands and to maximize expressive functions through the use of her hands.

Her verbal expressive abilities handicapped and severely limited her educational achievements, which could be considered borderline defective, with reading, spelling, and arithmetic at the first grade level (Educational Quotients = 70, 67, and 73, respectively).

M.M.'s psycholinguistic functioning indicated an overall retardation which was well in keeping with her overall intellectual functioning (Language Age = 6 years, 2 months, except for her auditory input which was above her age level (8 years, 10 months) and her visual association (8 years, 3 months). This pattern was similar to what she demonstrated on all other tests summarized above. Although her perceptual functioning was also retarded (Frostig Perceptual Quotient = 68), nonetheless, her knowledge and ability to deal with spatial relations through the use of eye hand coordination remained one of her best areas of functioning (7 years, 6 months). Since other visual functions were handicapped and this modality showed a great deal of discrepancy in functioning, one would suggest its thorough evaluation through appropriate referral to rule out any gross visual defect.

The retarded educational functioning and the discrepancy in intellectual functioning affected her emotional adjustment, especially in the way she viewed herself as a small, rather deprived little girl who needed more attention than she felt she was getting. Her self concept, therefore, was similar to that of a handicapped child although outwardly she did not appear to be one. She viewed herself as lacking physically as well as emotionally. Other components of her emotional immaturity were passivity and an inability to deal with human figures. This withdrawal from human contact perhaps was due to her educational failures and the feeling of failure that had been produced during her brief schooling.

On the basis of these results it was recommended that educational placement take account of her assets as well as her liabilities. Teaching should utilize her major input channel, which was hearing, maximizing the eye hand coordination which seemed the best of her expressive assets. Visual input and verbal expression as an output should be avoided, since these seemed to be the worst liabilities in her overall functioning. By maximizing the feeling of success and mastery through the use of her sensory motor assets, one would hope that her feelings of inadequacy and of failure would be decreased. It was important for this girl to feel that she was successful in a few areas, with success hopefully spreading to other more deficient areas of functioning. It was suggested that perhaps teaching should take the form of records played for auditory input, while her levels of understanding and of learning would be fostered through manual activities rather than through verbal or silent reading. Her skill in writing could be increased by actual manipulation of letters and molding of objects.

On the basis of our evaluation an EEG was recommended with an accompanying letter to the originally referring physician suggesting that a "highly speculative guess at this point would indicate a mild localized dysfunction, possibly bilateral but with greater lateralization in the left hemisphere." Unfortunately, the EEG report was not as specific as we would need to have it. "The recordings were made with 21 scalp electrodes in standard placement; cooperation was good throughout. The finding was of a basic record of irregular 9-10/second activity of varying but mostly higher amplitude with some 4-7/second activity intermixed. Though such activity was generalized, it had a prominent posterior locus. A considerable buildup occurred during hyperventilation. A similar intermittent buildup occurred with photic stimulation, though the driving response was of a more normal appearance. Recordings made during a period of spontaneous sleep revealed normal sleep activity. The impression was one of an abnormal record with generalized irregularities of the background performance. This was felt to be a dysrhythmic but not true epileptiform performance."

Six months after this evaluation and recommendations we received the following letter from M.M.'s teacher:

> I would like to inform you concerning the progress of [M.M.] and to thank you for your report to me. I have tried to carry out as many of your recommendations as possible. I find that one-to-one instruction with young children is always more effective. However, I have ordered some records for the teaching of phonics that I hope will prove to be helpful.
>
> [M.M.] has been making steady progress, slowly of course, but with real effort on her part. She has been dictating her stories to me then copying them in her own book. She seems to respond to a kinesthetic approach to some words and is reading material with a vocabulary of primer difficulty.
>
> She is able to make up her own stories with help in spelling. I am enclosing one of my favorites. Her manuscript is excellent as you can see.
>
> Our school has gone into an ungraded setup for next year and [M.M.] will go on to a second year room with her class. She will be able to progress at her own rate and will be made comfortable doing so. I feel very encouraged and hope I have told you some of the things that are pertinent.

On about the same day her mother reported:

> We are sending her to a day camp for the next two weeks to improve her swimming. We feel she has shown a great deal of improvement at home and at school since January. She quit complaining about headaches and going to school. She has to have a good deal of help in reading but now she wants to read—previously the desire was not there.

The parents brought her back for reevaluation, as we expressed the wish to follow her up after an outline of our program was verbally explained to them. The results of the second evaluation, after one year and a half, indicated the unreliability or, at least, the variability of our tests.

TABLE 4

Case Study 1b Summary Sheet—Psychological Evaluation for Use with Children: 6 years 0 months to 8 years 11 months

Name: M.M. Sex: F Age: 9-10.

		MA	*IQ*
1. Draw-A-Person (DAP)	Boy	5-9	58
	Girl	6-3	63

TABLE 4—*Continued*

2. Peabody (PPVT)		6-10	74
3. R—L Discrimination			
No. Correct/Total=17/32			
R: 17 L: 17			
4. Frostig			
		Age Equivalent	*Scaled Score*
Eye Motor Coordination		10+	10
Figure Ground		8-3	10
Form Constancy		6-3	6
Position in Space		6-3	6
Spatial Relations		8-3	10
Total		PQ	84
5. ITPA			
		Language Age	*Scaled Score*
Auditory Decoding		8-10	1.30
Visual Decoding		8-9	.34
Auditory-Vocal Association		6-10	−1.25
Visual-Motor Association		7-10	−.44
Vocal Encoding		5-1	−1.69
Motor Encoding		7-4	−.44
Auditory-Vocal Automatic		6-10	−1.65
Auditory-Vocal Sequence		4-4	−2.54
Visual-Motor Sequence		5-1	−2.01
Total		6-8	−2.13
Benton C=+2 L=3			
E=−4 R=5			
6. Wide Range Achievement			
	Grade	*EA*	*EQ*
Reading	1.7	6.7	66
Spelling	1.3	6.1	61
Arithmetic	1.2	6.0	60
7. WISC			
Information	4	V. IQ 70	
Comprehension	7	P. IQ 86	
Arithmetic	3	Full IQ 75	
Similarities	5		
Vocabulary	7		
Vocabulary IQ	81		
Digit Span	5		
Coding	—	Ratios	
Picture Completion	8	1.———	

TABLE 4—*Continued*

	Picture Arrangement	5	2.———
	Block Design	9	3.———
	Object Assembly	10	
	Mazes	—	
8.	MCPS		
		Raw	*Norms*
	Conformism	—	—
	M — F	—	—
	Maturity	—	—
	Aggression	—	—
	Withdrawal	—	—
	Hypermotility	—	—
9.	Rorschach ———————		
	Supplementary Tests		
10.	Family Relations ———————		
		Time	Cred.
11.	Trail Making A	———	———
	B	———	———
12.	CAP		
		MA	*IQ*
13.	Stanford-Binet	———	———
	S-B Vocabulary	———	———

There were decreases in the drawings (DAP), PPVT, WRAT, and WISC. The only increase was in perceptual functioning (Frostig) and Benton's Right-Left Discrimination. However, the relative inferiority of verbal below the visual-motor functioning, as found in the first evaluation, remained consistent.

This case as well as the following not only indicate all of the many subtle relationships among cerebral dysfunctions, educational achievement, and emotional adjustment, but they also illustrate the ease with which the laboratory method allows reevaluation after a period of rehabilitation. However, if one person has to do the testing and retesting alone this luxury would be very difficult to undertake. With the help of a technical assistant, one is able to reevaluate any child.

Case Study 2

B.C. evidenced a clear cut visual motor deficit. This deficit was not only responsible for his inability to write, but it also produced in him, secondarily, a great deal of hostility, negativism, and hyperactivity. These reactions appeared to result from strong, underlying feelings of personal inadequacy and even from depression.

TABLE 5

Case Study 2a Summary Sheet—Observations of Child

Name: B.C. Sex: M Age: 6-11

Describe child (physically): Height and weight seem typical for his age. No obvious hearing problems. When holding a pencil and attempting to write, his hand shows a slight tremor, and it is a fight for him to control the shape of the letters he forms—bears down heavily and is exhausted quickly.

Describe his behavior: Quiet at first, then he begins looking at the clock and letting E. [examiner] know when he has to go. He finds it very hard to sit in the chair for long and usually is up and down five or six times a period and more.

Note changes in behavior within each hour: Gets more impatient. "I think my Momma is calling me." "This is the last one. I'm not going to write any more."

Note changes in behavior between hours: The more his desire to terminate is blocked, the more hostile his behavior, not words, becomes, such as responding incorrectly to statements I feel sure he knows and when confronted with this will respond correctly, etc. Instead of pointing with his finger, uses elbow.

Eyes watery, once evidenced mucus. Maybe due to cold. M. [mother] says child was recently examined by pediatrician and given o.k. Eyes never been checked otherwise. Last day the WISC and inquiry of Rorschach needed to be completed. Although on previous visits B. voiced his displeasure at being here, he was never really more than quietly negative. The last session not only was he looking out the window every other minute, but he would switch chairs, slump in his chair, and push it toward the door—almost total refusal to put up with any more testing. As a last resort after all attempts at responding failed even though B. would stay in his seat, Mr. C. was asked to sit in for discussion if B. continued to refuse. Even a threatened spanking did little to move him. The E. had previously tried to explain why he was here, the purpose of the tests, etc.

On the WISC performance section, B.D. [Block Design], he would turn up blue, yellow, red blocks and say his looked just like an all red and white pattern. The M. was asked about color blindness and knew of none in her family. Later, to see if B. had faked his designs and color to terminate the test, he was asked again to make A and B [sample patterns]. He made A correctly and rather quickly. Although the colors were right for B., his design was wrong. The WISC and especially, the Performance section, I feel, cannot be considered representative.

How did the child relate to you during testing: Although B. would have liked to terminate and not complete assignments, with insistence and some control he

TABLE 5—*Continued*

would, even if he did not want to. He tried the limits constantly and has been uninterested in practically all the tests.

Note any unusual characteristic of the child's behavior: Child looks to E. for approval or disapproval of his answers looking for clues as to their rightness or wrongness —dependency fostered often in primary school children.

TABLE 6

Case Study 2a Summary Sheet—Psychological Evaluation for Use with Children: 6 years 0 months to 8 years 11 months

Name: B.C. Sex: M Age: 6-11

			MA	*IQ*
1.	Draw-A-Person (DAP)	Boy	5-3	73
		Girl	6-0	85
2.	Peabody (PPVT)		6-10	98

3. R—L Discrimination
No. Correct/Total=28/36
R: 14 L: 14

4. Frostig

	Age Equivalent	*Scaled Score*
Eye Motor Coordination	4-0	6
Figure Ground	6-0	9
Form Constancy	4-9	7
Position in Space	5-6	8
Spatial Relations	4-0	6
Total	PQ	72

5. ITPA

	Language Age	*Scaled Score*
Auditory Decoding	8-10+	1.21
Visual Decoding	6-8	.02
Auditory-Vocal Association	7-3	2.11
Visual-Motor Association	7-10	.41
Vocal Encoding	5-1	—.68
Motor Encoding	5-5	—.33
Auditory-Vocal Automatic	7-7	.58
Auditory-Vocal Sequence	8-6+	2.80+
Visual-Motor Sequence	4-4	—2.07
Total		—.02

TABLE 6—*Continued*

6. Wide Range Achievement	*Grade*	*EA*	*EQ*
Reading	3.1	8-3	120
Spelling	1.9	6-9	97
Arithmetic	1.5	6-4	90

7. WISC		
Information	9	V. IQ 90
Comprehension	9	P. IQ 71
Arithmetic	10	Full IQ 79
Similarities	7	
Vocabulary	8	
Vocabulary IQ	—	
Digit Span	7	
Coding	4	Ratios
Picture Completion	11	1.______
Picture Arrangement	5	2.______
Block Design	4	3.______
Object Assembly	5	
Mazes	—	

8. MCPS	*Raw*	*Norms*
Conformism	23	50
M — F	22	50
Maturity	13	50
Aggression	50	55
Withdrawal	18	54
Hypermotility	6	50

9. Rorschach ______________________

Supplementary Tests

10. Family Relations ______________________

11. Trail Making	Time	Cred.
A	______________	______________
B	______________	______________

12. CAP

13.	*MA*	*IQ*
Stanford-Binet	______________	______________
S-B Vocabulary	______________	______________

B.C.'s visual motor handicap was consistent in all areas of functioning. Intellectually, for instance, his handicap brought about a wide discrepancy between his low average verbal functioning (Verbal IQ = 90) and his borderline defective visual motor problem solving (Performance IQ = 71); on the other hand, there was some evidence to indicate that at least his visual verbal functioning was within average limits (Peabody IQ = 98). Indeed, this discrepancy in functioning was even better illustrated by his educational achievements. Educationally, his reading was superior (Reading Quotient = 120), but when his reading was compared to spelling and arithmetic, his functioning decreased to average and low average levels, respectively (Spelling Quotient = 97; Arithmetic Quotient = 90).

These results were consistent with his visual motor performance on the Frostig (Perceptual Quotient = 72). The basis for the foregoing discrepancies in motor functioning lay especially in his psycholinguistic abilities (ITPA). Here, he revealed a pattern of superior functioning in auditory decoding (auditory vocal associations and auditory vocal sequencing); while his visual decoding, although average, became more inadequate whenever it was linked to other expressive modalities, such as vocal or motor encoding. Whenever the visual modality was coupled to motoric expression, this aspect of functioning became relatively incapacitated. This discrepancy in psycholinguistic functioning, therefore, was also consistent with his near average verbal functioning and with his defective visual motor functioning.

In speculating about his aggressive hostility, negativism, and hyperactivity evidenced during testing, it was felt that these actions were more of a reactive nature to the threat of being tested and being evaluated rather than persistent personality traits. This child was hitting back perhaps because of tremendous feelings of inadequacy produced by his visual motor deficit and other factors. There was no question that his feelings had suffered a great deal. He had developed a self picture of an inadequate, almost worthless, abandoned child. These feelings, therefore, may have been the basis for his overreaction to threat. Any threat that might have augmented his feelings of inadequacy might have produced anger and hostility. In turn, these feelings also had a great deal to do with his intellectual performance during testing, since his lack of cooperation did not allow a valid evaluation of his actual or potential functioning. Probably the scores reported here may represent the very minimum effort he was willing to exert.

On the basis of these results, therefore, it was recommended that any teaching procedure would need to exploit the auditory vocal rather than the visual or visual motor modalities. In other words, the emphasis should be on his specific assets in the auditory area rather than on the visual and visual vocal area thus augmenting his feelings of failure. By so doing, some of his feelings of inadequacy and subsequent hostile overreactions to external threat might disappear. Some therapeutic support and attention through play might be given him in trying to bolster his depleted self esteem. In terms of his visual motor liability, he could be helped through calisthenics, physical exercises, and sports.

The program of rehabilitation for B.C. on the basis of his initial evaluation was a multiple one. He had to be moved to a special school where, however, the principal insisted on his being kept under sedation by the referring pediatrician. It was planned to see him for play therapy with occasional conferences with the parents. The mother did not feel that any therapy for herself was necessary. She viewed and reported herself (verbally as well as on the Leary Interpersonal Checklist which is administered routinely to all mothers) as a very adequate mother, except for "blind spots" in her feelings surrounding B.C.

B.C. was seen for twenty sessions during a seven month period. Both parents were seen for conferences at the beginning, in between, and at the end. In addition, B.C.'s mother was seen for brief conferences occasionally and kept in touch by telephone. After a few months, she related aspects of B.C.'s birth that were unknown at the beginning. Initially, she had reported that B.C. had been severely burned at two months of age by hot water from a vaporizer. His entire right side was burned, and he was kept in a hospital for two months, initially in critical condition.

The mother was unable to express any feelings about this incident and denied having treated this child any differently than any other. Later on, however, she related that he was born a few days after the death (from cancer) of a six year old brother. At this point, she was able to acknowledge and to express a great many feelings that she had repressed in the intervening years. She admitted a great deal of overprotection for B.C. that she had not shown for his other healthy brothers and sisters.

B.C. reacted strongly to the special school where children with evident physical handicaps made him face a reality he had rejected. He was not visibly handicapped. In fact, he was a handsome blond child who attracted adults' admiring attention wherever he went. He behaved so poorly in school that he had to be moved to another school for emotionally disturbed children with no physical or organic handicaps. He adjusted to this new school well, and he is planning to continue there at least for another year until his spelling and arithmetic improve.

The following are the results and interpretations of B.C.'s retesting at the end of play therapy.

TABLE 7

Case Study 2b Summary Sheet—Psychological Evaluation for Use with Children: 6 years 0 months to 8 years 11 months

Name: B.C. Sex: M Age: 7-8

		MA	*IQ*
1. Draw-A-Person (DAP)	Boy	6-6	83
	Girl	6-6	83
2. Peabody (PPVT)		8-3	102

3. R—L Discrimination
No. Correct/Total=27/32
R: 22 L: 24

4. Frostig

	Age Equivalent	*Scaled Score*
Eye Motor Coordination	4-6	6
Figure Ground	5-3	7
Form Constancy	7-0	9
Position in Space	5-0	7
Spatial Relations	4-0	5
Total	PQ	66

TABLE 7—*Continued*

5. ITPA	*Language Age*	*Scaled Score*
Auditory Decoding	7-1	—.24
Visual Decoding	8-9	1.29
Auditory-Vocal Association	7-8	.02
Visual-Motor Association	7-10	.05
Vocal Encoding	4-5	—1.68
Motor Encoding	5-5	—.98
Auditory-Vocal Automatic	8-9	.89
Auditory-Vocal Sequence	8-6	2.33
Visual-Motor Sequence	4-7	—2.01
Total	7-5	—.32

6. Wide Range Achievement	*Grade*	*EA*	*EQ*
Reading	3.8	9-0	116
Spelling	1.7	6-7	84
Arithmetic	1.2	6-0	76

7. WISC		
Information	8	V. IQ 95
Comprehension	13	P. IQ 78
Arithmetic	6	Full IQ 85
Similarities	6	
Vocabulary	10	
Vocabulary IQ	100	
Digit Span	12	
Coding	5	Ratios
Picture Completion	7	1.———
Picture Assembly	8	2.———
Block Design	7	3.———
Object Assembly	7	
Mazes	—	

8. MCPS	*Raw*	*Norms*	
Conformism	—	—	
M — F	—	—	
Maturity	—	—	
			Invalid
Aggression	—	—	
Withdrawal	—	—	
Hypermotility	—	—	

9. Rorschach ————————————

TABLE 7—*Continued*

Supplementary Tests

10. Family Relations ____________________

		Time	Cred.
11. Trail Making	A		
	B		

12. CAP

	MA	IQ
13. Stanford-Binet		
S-B Vocabulary		

B.C. marched into the testing room with confidence. He appeared even a little haughty. He was very correct, referring to the examiner as "Mr.," and he was assertive about the physical arrangements of the testing. He moved the chair and fixed things to make himself more comfortable. He became quite active during the testing. He finally elicited a mild reprimand from the examiner to leave other materials alone and to pay attention to the materials at hand. He was efficient, very verbal, but neither tense, withdrawn, nor negative. He was just rather aggressive, but he did respond to corrections about his exuberance.

Quantitatively, B.C.'s functioning still showed the differential effects of cerebral dysfunction in the visuomotor area. Verbally B.C. still functioned within the average limits (WISC Verbal IQ = 95, Vocabulary IQ = 100, and Peabody IQ = 102). However, his visuomotor functioning was still in the borderline defective category (Frostig Perceptual Quotient below 66, WISC Performance IQ = 78, and DAP IQ = 83).

His differential pattern of intellectual functioning affected his educational achievements a great deal. His reading was still in the superior range (Educational Quotient = 116), but his spelling and arithmetic were still relatively retarded (Quotients 84 and 76, respectively). His reading was on the third grade level, while his spelling and arithmetic were still on the first grade level. These particular discrepancies in functioning were related to his psycholinguistic abilities, especially in complex visuomotor sequencing which was low. He appeared more adequate on tasks involving auditory-vocal modalities, a pattern which was consistent with his verbal and reading superiority.

His overall emotional adjustment could be characterized as varying between a passive and aggressive expression of his impulses, with heavy emphasis on aggression and fighting. There was still a good deal of distortion toward the parental figures who were viewed as rather threatening to him. He was unable to express any outward hostility against them.

In terms of the differences between pre- and posttherapy evaluations, the quantitative results were very much the same. B.C. remained a child of average to bright average ability and potential with definite handicaps in the visuomotor area and some areas of learning, particularly spelling and arithmetic. This discrepancy might derive from his early physical trauma. The most noteworthy change that occurred was a much greater degree of articulation in his emotional reactivity and absence of depressive symptoms which were present in the previous testing, as shown in Table 8. The depressive feelings which were strongly present in the pretherapy testing gave place to a greater ability to express much more articulately aggressive responses with a lesser degree of perseveration. Table 8 contains a comparison of his Rorschach free associations.

TABLE 8

Free Rorschach Associations of B.C.

Cards	*Before Play Therapy*	*After Play Therapy*
I	cloud	devil's ear and bat
II	pussy cat	some kind of fly
III	plain old cloud	an animal—2 poodle dogs
IV	plain old cloud	gorilla dancing
V	butterfly	butterfly
VI	nothing	cat laying down dead
VII	rabbits up in the air	4 headed dragon on rocks
VIII	2 raccoons climbing a tree	bears climbing a tree
IX	2 bears scaring	2 animals fighting
X	2 horses looking at those green things	some lobsters—all sorts of water animals climbing on those

Scored according to Cleveland and Fisher's body image scheme, his Rorschach responses resulted in six penetrations of barrier responses before therapy and none after therapy.

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Part 4:
Approaches to Treatment

Minimal Brain Dysfunction: A Dilemma for Educators

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Children with minimal brain dysfunction have been receiving more attention recently than at any other time in the special education movement. Credit for initiating a focus on this syndrome goes to Strauss, who attached the term brain injured (or exogenous) to this group of children twenty-one years ago (Strauss and Lehtinen, 1947). One can only speculate on the reasons for the heightening concern since then in this area. Probably these three factors are operating: (a) the increased number of such children today, (b) the growing sophistication and outspokenness of parents, and (c) the dissatisfaction of parents and professionals alike over placing such children in either the regular grades or in special classes for the retarded. In any event, parents and teachers are about at their wits' end, and are demanding that professional persons increase their efforts to ameliorate the many problems which these children present. And they have hardly begun to make their voices heard. Community pressure is increasing to establish special school programs specifically for this group. New parent groups are becoming organized to the point where they can exert the kind of pressure that other groups have done in the immediate past on legislators, on school board members, and on other elected officials. Even professional persons—in addition to educators—are beginning to feel the surge of reproach and demand. The critical needs of these children require that we focus our attention on this area with new vigor and imagination. This article explores this issue from the point of view of the educator.

Describing and Defining the Syndrome

Most of us who work with such children believe we have a fairly clear picture in mind of the syndrome. The following would be a typical sketch.

> Ed is a ten year old boy without a motor handicap other than clumsiness. His speech is fairly distinct. Ed's intelligence quotient is 80. His teacher complains that he talks incessantly, constantly interrupting the class with irrelevant remarks. He has a strong need for attention and thus bothers other children who are working by knocking things off their desks, hiding their pencils, and hitting them on the head. His learning patterns are very uneven. It is nearly impossible to settle him down to academic work because he cannot concentrate on one thing for any length of time. In fact, he is at the mercy of any idea which occurs to him, or of any environmental event which reaches him.

One would think that these children would be easy to define and identify. Such is not the case. For example, rather recently, clinical teams of a chain of child study centers located across a southern state were meeting together as a group for the first time. In studying the annual reports of these dozen centers, they discovered that in some clinics, 30 to 40 percent of the children seen during the year were diagnosed as emotionally disturbed, and 10 to 20 percent were placed in each of the categories of mentally retarded and brain injured. In other centers, 30 to 40 percent were labeled mentally retarded and relatively few were labeled brain injured and emotionally disturbed. In still other centers, 30 to 40 percent were diagnosed as brain injured, with much smaller numbers placed in the categories mentally retarded and emotionally disturbed. (Other children seen fell in other areas of exceptionality.) Now how could this be? When presented with these data, the clinicians discussed them at length. They contended that part of the variance could be accounted for by the type of service being initiated in a community, thus leading to selective referral. Another explanation was that most children had all three types of disability and the conflict arose in designating the major one. However, another factor seemed more crucial. The diagnostic category selected depended on the training, professional makeup, philosophy, and predisposition of the diagnostic team. Three different groups, depending on their biases, could label the same child brain injured, emotionally disturbed, or mentally retarded. A dilemma indeed!

Similar findings are revealed by a review of psychological research literature. Many psychological tests have been devised to elicit different responses among the organically impaired, mentally ill, and mentally retarded. Among these are the Goldstein-Scheerer sorting tests, the Graham-Kendall Memory-for-Designs Test, the Bender Visual Motor Gestalt Test, marble-board tests, the Halstead and Reitan batteries of neuropsychological tests, and many others. Such critical reviews of the research literature as those by Yates (1954), and Klebanoff, Singer, and Wilensky (1954) point out that such tests usually differentiate any and all groups of the organically impaired, mentally retarded, and mentally ill from the normal. However, their record for differential diagnosis among the three groups is appallingly poor. It is even worse for individual cases.

Neurological findings have been equally equivocal. In general, the neurologists, pediatricians, or pediatric neurologists report that they can find no abnormal signs in many of these children who are being labeled minimally brain damaged. In many cases, the EEG patterns are normal; however, in retrospective studies comparing groups of such children with normal controls, the former usually have significantly more abnormal EEG's and neurological signs. But this is of limited value in individual diagnosis. A puzzling state of affairs indeed!

At this point in our knowledge, the question arises as to the utility of labeling such children. On the one hand, there are those who contend we should get rid of all labels in this area. They base their arguments on such positions as the following: (a) the labels we have devised, to date, are not badges of distinction; (b) children in this category are so different from one another that no group treatment is known to be effective; and (c) labels are blocks to thinking. On the other hand, there are those who contend we must find and agree on a label. Their arguments include the following: (a) we usually cannot provide special education services, unproven as they may be, until we describe the children we propose to serve; and (b) we cannot advance knowledge in the area until we describe the condition. In any event, there has been no paucity of terms introduced. Some of these descriptive terms are:

agenesis child
birth damaged child
chronic brain syndrome
clumsy child
exogeneous child
hyperactive child
hyperkinetic syndrome
imperceptive child
interjacent child
major learning disordered child
minimal cerebral injured child
minimal brain dysfunctioned child
nervous child
neurologically handicapped child
neurophrenic child
neuropsychologically impaired child
neurosensory disordered child
organically impaired child
perceptually impaired child
perceptual-motor impaired child
psychomotor disordered child
psychoneurologically disordered child
Strauss syndrome

As one hears these terms, besides being confused and dismayed, he may also tease out four clusters. One group presupposes an organic etiology, for example, in the term minimal brain damaged. A second group resorts to behavioral terms with no attempt to imply causation, such as hyperactive, perceptually impaired, or learning disordered. A third group uses a neutral term, such as the "Strauss syndrome." A fourth group straddles the fence on etiology, implying an interaction of environmental and biological factors—for example, "psychoneurological learning disorders."

Where is the field moving in terms of consensus on terminology? There are two developments.

First, the National Institute on Neurological Diseases and Blindness of the US Public Health Service and the National Society for Crippled Children and Adults have collaborated in sponsoring a task force on terminology and identification. Clements (1966) has prepared a report on the deliberations of this group which chose the term *minimal brain dysfunction* to describe the syndrome. Since this task force was made up primarily of physicians, one can understand the proclivity to choose a term which implies causation, because this has generally been the route to prevention and medical treatment. The term brain dysfunction rather than brain injury leaves open the possibility of a biochemical malfunction, as well as trauma and inadequate development of neural structures. The biological scientists appear to be moving through the progression from birth injured to brain injured to biochemically disordered. The utility of this term for educators (if not for all behavioral scientists) remains to be demonstrated.

Secondly, the term, pupils with *learning disabilities*, has been selected through recommendation of representatives from professional organizations and special educators working with the Terminology Compatibility Branch, National Center for Educational Statistics, in the US Office of Education. Use of this term is suggested in a handbook being developed for use in pupil accounting by local and state school systems. As defined, the term includes the Strauss syndrome child, and others who show (a) an imbalance in cognitive development, and (b) a marked underachievement. (For reporting purposes, children with learning disabilities are identified also according to other characteristics, e.g., gifted, partially seeing, retarded, or within normal range.) For educators, this term has two advantages: it does not imply causation, and it indicates a need for special education. Thus, while it is a very loose term, it may have utility in special education.

For purposes of behavioral research, a more restrictive term than either minimal brain dysfunction or learning disability, which would be amenable to an operational definition, would appear to be needed. My personal preference is for the neutral term *Strauss syndrome*, a term advocated by Stevens and Birch (1957). In any event, the movement toward concern—if not consensus—on terminology is a heartening one. Whether one chooses the term minimal brain dysfunction, learning disability, or Strauss syndrome, it is generally agreed we are often talking about the child so skillfully described by Strauss in 1947.

What is the body of knowledge on the behavioral correlates of this condition? What procedures have been advocated for the education of these children? Three sections follow dealing with these issues: first, the theoretical-clinical literature is summarized; second, the educational approaches are advocated; third, some empirical evidence on their effectiveness is presented.

Theoretical-Clinical Literature

The initial impetus to the field appears to have come from the classic research by Goldstein and Scheerer (1941) on traumatically brain injured World War I soldiers. Their major contributions included the following: (a) they shifted us back from a localization theory of brain injury to thinking about generalized brain damage; (b) they gave us the terms "abstract" and "concrete" thinking, and characterized the brain injured as concrete thinkers; and (c) they gave us four characteristics of the brain injury syndrome in adults—catastrophic reaction, rigidity, distractibility, and, again, the concrete mode of thinking.

The next links in the chain are the contributions of Strauss, Lehtinen, Werner, Kephart, and others in relating (or a better term would probably be extrapolating) Goldstein's findings on brain injury from adults to children. As the leader of this group, Strauss described his subjects as having four behavioral characteristics: (a) perceptual disorders, (b) perseveration, (c) thinking and conceptual disorders, and (d) behavioral disorders, especially including hyperactivity and disinhibition. He quite clearly noted that his three biological signs—slight neurological signs, a history of neurological impairment, and no history of mental retardation in the family—could be all negative, and still the child could be diagnosed as Strauss brain injured solely on the basis of the behavioral characteristics. It is most unfortunate that Strauss named his subjects brain injured. Workers in this field have not read the fine print in the Strauss-Lehtinen work, or talked with persons who worked with Strauss. Thus, too many have equated a Strauss type child with a neurologically impaired child. Generally, special educators today label a child as fitting the Strauss syndrome when he displays hyperactivity, incoordination, lack of inhibition, distractibility, and an uneven pattern of learning disabilities, especially in language. Perhaps disturbances of perception and concept formation should also be mentioned, but these are more difficult for teachers to observe. Educators recognize that these children do not possess these seven characteristics in the same amount; however, if the children demonstrate the first five to a considerable degree, the term "Strauss syndrome" is usually attached to them by teachers.

Another thread in the theoretical clinical literature comes from the Bellevue group in New York City. Schilder (1935), Bender (1956), and others have argued that specific lesions of the brain result jointly in specific disabilities in the motor and psychological areas. Body image has a major place in this dual disorganization.

Gellner (1959) and her associates at the Columbus (Ohio) State School have identified four classifications of so called brain injured children: (a) visual somatic, (b) visual autonomic, (c) auditory somatic, and (d) auditory autonomic.

Doman, Spitz, Zucman, Delacato, and Doman (1960) have a theoretical rationale for an unusual educational treatment. They argue that the organization of the brain progresses developmentally from the medulla to the midbrain up to the cerebral cortex. Brain injury results in neurological disorganization which can be remedied by taking the child through the progression of neurological organization moving from primitive to more complex forms of perception and movement.

Benoit (1960) has applied Hebb's neuropsychological theory to the field. Hebb postulates that sensory and motor behavior is composed of cell assemblies. Congenital brain damage, before the cells have assembled, is more serious than adventitious brain injury which occurs after the pathways and higher mental processes have been established. The latter is not so disorganizing because, once concepts have been formed, they are less dependent on specific nerve pathways. Such brain injury may not eradicate old learnings but will interfere with new learnings.

The Halstead (1947), Reitan (1962), and Luria (1961) literature promises to have a profound effect on the education of so called brain injured children. These men have been primarily interested in studying the effects of brain lesions on behavior. In doing so, they have devised psychological diagnostic tests to determine the location of brain lesions in adults. Researchers are presently modifying their psychometric instruments for use with children, possibly yielding valuable dividends in providing educators with a profile of behavioral characteristics of individual Strauss-type children.

Kirk and McCarthy (1961) have developed the Illinois Test of Psycholinguistic Abilities, yielding a profile of nine measures of oral language with norms for children two and a half to nine years of age. This is the first major thrust at providing a standardized diagnostic instrument upon which to base remedial instruction procedures.

Frostig, Lefever, and Whittlesey (1961) have taken the position that neurological damage in children results in severe visual perceptual disturbances which need to be treated by systematic training.

A final theoretical position about the nature and modification of human behavior bears mentioning—the Skinnerian (1953) one. This position would contend that concern for the etiology or behavioral characteristics of the Strauss syndrome is not a major consideration in modifying behavior. Furthermore, supporters of this theory would advocate an individualized approach rather than advocate attempts to group children with common characteristics into clusters with similar labels. Finally, they would agree that it is possible to teach almost anything to almost any child by programing instruction into small, sequential steps, and reinforcing (rewarding) appropriate behaviors.

Education Approaches

The various educational approaches based on the theoretical-clinical literature just presented are outlined below.

Psychomotor Development. One of the educational approaches for the Strauss type child stresses physical education, motor development, motor training, mobility, coordination, or psychomotor development. Such an emphasis is a part of at least three approaches—the Doman-Delacato, Kephart, and Schilder-Bender positions. The Philadelphia group under Doman and Delacato (Doman, et al., 1960) see the so called brain injured child as needing to move through seven stages of mobility: (a) movements of the arms and legs without bodily movement, (b) crawling in the prone position, (c) creeping on hands and knees, (d) walking with the arms used extensively in balance, (e) walking with the arms not necessary for balance, (f) walking and running in different patterns, and finally (g) using the hands and legs to perform tasks other than those simply involved in mobility. Kephart and his group at Purdue University also stress the need for systematic training to foster motor development among the brain injured. Since motor development proceeds from the head to the feet, he argues that motor training needs first to involve the muscles of the head, and then should proceed downward to the arms and shoulders, to the abdomen, and finally to the legs and feet. Furthermore, he contends that motor development proceeds outward from the central axis of the body toward the periphery. Therefore, large movements of the arms and legs should precede fine movements of the wrist, fingers, ankles, and toes. He then proceeds to outline a wide variety of exercises to develop specific movements, arguing that the brain injured need special attention in the development and coordination of *patterns* of movement needed for complex acts. Kephart, like Doman and Delacato, argues that the brain injured need to be taken back and brought up through the different stages of motor development to establish these complex motor movements involving balance, coordination, and movement; otherwise, the child is likely to develop splinter skills. Kephart stresses the need to develop laterality, including an awareness of the difference between the right and left sides of the body. Friedus (1964) has outlined a variety of techniques for developing body image, based on the Schilder-Bender constructs. For example, she includes feeling and naming parts of the body, counting on the body, and crawling under bars. Thus we see a remarkable overlap among the recommendations for psychomotor development among the Philadelphia, Purdue, and Bellevue groups. While this emphasis does not constitute a total program of training for the brain injured child, some authorities obviously consider it quite basic. Thus, for the young child or the severely handicapped youngster, these persons believe that major attention must be given to this psychomotor, or first stage.

Perceptual Training. The second area (or stage) of training for the so called brain injured child, perceptual training, shows up in one form or another in many of the rationales. This may be as narrow as training in visual perception to as broad as a comprehensive program of sensory motor training. After a child has developed an adequate level of motor proficiency, Kephart argues that the training should shift to the development of perceptual organization. He considers it extremely important to match perceptual data with motor activity through developing exercises so that visual and auditory information is integrated with the tactual system. Thus he argues that perceptual learnings depend upon prior motor learnings. When a child is ready for it, Kephart recommends pencil and paper exercises to develop visual perception and left to right orientation, so necessary for reading readiness. The Los Angeles group headed by Frostig (1961) focuses on visual perception for the neurologically handicapped child at the stage at which this can be developed through pencil and paper exercises. She has published a visual perception test, and a pencil and paper program for training in areas of specific visual disabilities. Only because of the need for brevity here additional authorities who emphasize the need for perceptual motor training will not be cited. In fact, so many writers focus on this area that it is not surprising that classes established for perceptually impaired children enroll youngsters with characteristics very similar to those of children in classes designated for brain injured children.

Concept Formation. A third stage (or emphasis) in training programs for the brain injured focuses on concept formation, including training in the various school subjects. Here the pioneer work was done by Lehtinen and is reported in Part 2, Volume 1, of *Psychopathology and Education of the Brain Injured Child* by Strauss and Lehtinen (1947). Recommended guidelines for the instruction of the pupil with the Strauss syndrome included the following:

1. A nondistracting school environment should be provided. Translucent rather than transparent window panes should be used in the classroom. The teacher's dress should be plain and free from ornaments. The class should be located on the top floor and made free from distracting stimuli. Cubicles and screens should be utilized to reduce distractions.
2. Instruction should be individualized. The class groups should be small, with 12 children as a maximum. For individual work, pupils should be removed to the periphery of the group, faced toward a wall, or screened off from the rest of the children by the cubicles.
3. An elemental rather than a global approach to teaching should be emphasized. For example, the teaching of reading should begin with the learning of individual letters; later these should be assembled into words; and finally the words should be used in sentences, paragraphs, and stories.
4. Emphasis should be placed on the use of colored letters, words, and numbers as well as other concrete cues to focus the child's attention on the relevant materials.
5. Motor activity should be involved in academic learning, with emphasis on concrete manipulative materials.
6. Emphasis should be placed on the basic tool subjects. Instruction in social studies, geography, and science should be considered incidental.

7. No use should be made of the project or unit method.
8. Social activities, group learning, and oral language should be deemphasized.

This is but a brief glimpse of the Lehtinen techniques, which have had almost universal acceptance by educators implementing special education programs for the Strauss type child. In the next section, some research will be presented on the efficacy of these Lehtinen techniques. Kephart, as one of the Strauss-Lehtinen group, utilizes these techniques. He contends that solid concepts rest upon solid percepts which in turn rest upon solid basic motor patterns. However, he deemphasizes concept and symbolic learnings, arguing that educators are preoccupied with this area and tend to move on to it before the child is adequately trained in the motor and perceptual areas.

Epps, McCammon, and Simmons (1958) have developed teaching techniques for the four types of so called brain injured children described by Gellner (1959). For the visual somatic type who has movement blindness, a kinesthetic and auditory approach, combined with a blindfold, is recommended. For the visual autonomic type who has a visual disability that is close to total blindness, it is necessary for the individual to learn about his environment through tactual and auditory techniques. For the auditory somatic type, who has sound and word deafness, it is necessary to use a visual approach. For the auditory autonomic type who has meaning deafness, it is important to stress meanings, in that these children are often very verbal but the materials are incomprehensible. This group tends to stress teaching to strengths. Very clearly, the Gellner-Epps techniques presented here might have been presented under perceptual training. Similarly, the Doman-Delacato approach outlined above under motor development may have been included here. Thus a total training program moves a child from motor to perceptual to conceptual training.

The other approaches to education for the brain injured child do not fit neatly within the three categories above, but do bear mentioning. The Strauss group, including Lehtinen, were concerned with modifying the unacceptable behavioral characteristics of the Strauss type child.

This group has argued that hyperactivity may be reduced by cutting down on environmental stimulation. The catastrophic reaction was to be controlled by keeping the brain injured child in a standard familiar environment and not placing him in situations of unusual stress. Thus the simplified, protective, routine environment was aimed at making these children more tractable. Some evidence will be introduced later to describe what happens to a hyperactive child in a barren and unstimulating environment. Perhaps drug therapy, including the use of tranquilizers, should be included here as a method of controlling the behavior of the Strauss type child.

Another approach to training is that of Kirk and Bateman (1962) discussed previously in connection with the Illinois Test of Psycholinguistic Abilities. These scientists believe that each individual child has his own unique profile of abilities and disabilities. They are systematically moving ahead to finding ways of teaching to the weaknesses of children so as to strengthen them. A recent volume elaborates on this approach (Kirk, 1966). The next decade is likely to see a good supply of research devoted to answering the question concerning the desirability of teaching to strengths, to weaknesses, or across the board when dealing with handicapped children, including the so called brain injured. Dunn, Smith, and Horton (1968) have developed Levels P, 1, 2, and 3 of the Peabody Language Development Kits aimed at an overall approach to oral language development, in contrast to the Kirk and Bateman (1962) approach of teaching to weaknesses.

Finally, under educational approaches, one must return to the Skinnerian position described previously. This group would see no need for the psychometric approach involving pre- and posttests, with a training program intervening. Instead, they would take the individual child forward, through programed instruction, from the point to which he has developed. Here, the method takes precedence over the characteristics of the child, classroom organization, or materials; however, it could be argued that the Skinnerian techniques of operant conditioning could be applied to the procedures advocated by all of the other writers mentioned above.

Empirical Evidence

Fragmented though it is, there is a growing body of knowledge concerning the behavioral correlates of brain injury. Good analyses of the literature have appeared recently in Robinson and Robinson (1965) and in Ellis (1963). No attempt will be made here to review comprehensively the literature on the more psychological aspects of the area. However, before becoming a devotee of any of the theoretical clinical positions which have been advocated, one should study some of these references, since research usually can be found to refute each of the various positions. Three or four examples are cited by way of illustration.

Hyperactivity has been regarded as a very consistent and stable symptomatic characteristic of the Strauss type child. Lehtinen has argued that hyperactivity is heightened even further by a stimulating environment and reduced by a barren one. Furthermore, it has been argued that the Strauss type child will learn better in a barren environment than in one of reasonable stimulation. These generalizations cannot be substantiated conclusively from the literature. While there are data to support this position, a review of this field by Cromwell, Baumeister, and Hawkins (1963), has uncovered contrasting theories and findings. The Strauss-Lehtinen group argue that incoming stimuli activate the brain and produce increases in body activity, and that reduction of external stimulation reduces the activity of the brain and the corresponding physical activities. A contrasting theoretical position is that the neural connections of brain injured children are less developed than are those of other subjects; hyperactivity on the part of brain injured children is an attempt to induce more stimulation. From this theoretical position, it would be predicted that increases in visual and auditory stimulation should decrease the activity level of individuals with sensory impairment, such as Strauss children have. Cromwell and his associates have conducted a series of studies on the effects of this stimulus variable. Generally, they have found that visual stimulation does reduce activity level. Furthermore, the activity level of hyperactive mental retardates decreases significantly with such tactual stimulation as bouncing and handling. Thus there is some support for the theoretical notion that hyperactivity may be a result of a seeking of stimulation because of partially blocked neural pathways. Burnett (1962) has one of the few studies investigating the influences of classroom environment on the school learning of retarded subjects with high and low activity levels. He measured the speed with which retarded subjects learned to read a list of words in both standard and restricted classroom settings, and found no significant differences, indicating that hyperactive children learn equally well in both types of standard environment. Evidence of this nature should lead us to be cautious in establishing a Lehtinen type classroom for hyperactive children. Conceivably, it may be the exact opposite situation to the one in which such children can learn best.

In terms of personality characteristics, again the research is far from uniform. Strauss and his coworkers have pointed out that most brain injured children display erratic, uncoordinated, uncontrolled, disinhibited, and generally unacceptable behavior. There are studies to support this contention; however, Semmel (1960) failed to find these personality characteristics more frequently among so called brain injured children than among mongoloid children of similar intellect. Zigler (1962) has argued that brain injured children may display these personality characteristics, not because of any central nervous system pathology, but because they usually come from middle and upper class homes where they are exposed to parents who display much more anxiety and who put much more pressure on the child than would be the case with the usual working class parents of cultural familial retardates, these factors causing them to have behavior disorders.

Another area of confusion deals with concept formation. Brain injured children are supposed to display greater variability in their abilities and disabilities. Yet we have a number of studies which do not support this observation. One such study was done by Capobianco (1956) who compared the arithmetic processes of cultural familial retardates and neurologically impaired subjects, some of whom displayed the Strauss syndrome. He found no differences in computation, reasoning, achievement, reversals, or understanding of the concept zero. Capobianco and

Miller (1958) analyzed the reading processes of the cultural-familial mentally retarded and the neurologically impaired, finding little or no difference between the two groups in their reading achievement or in their patterns of reading errors. Gallagher (1957) compared brain injured and non brain injured mentally retarded children on several psychological measures. Generally, he found no significant differences between the groups, except that children with central nervous system damage were superior in language traits. He concluded that having a physician identify a child as brain injured creates such a heterogeneous group that it is of little use to educators and psychologists. It must be pointed out that in the studies mentioned above, the researchers were dealing with neurologically impaired children, so identified by a physician. Thus they were studying brain injured children with biological signs, rather than children who displayed exclusively behavioral traits characteristic of the Strauss syndrome.

Turning to the effectiveness of special education for the brain injured, Gallagher (1960) conducted a three year experiment in a residential school setting, tutoring children of seven to nine years of age with neurological impairments. Each pupil was given one hour a day of individualized tutoring based upon that child's own pattern of strengths and weaknesses. It was a crash program of perceptual, conceptual, and language development exercises but there was no attempt to follow the Lehtinen approach per se. As contrasted to a control group, Gallagher's experimental subjects improved in intellectual development, increased in attention span, and achieved more in verbal than in nonverbal skills. He concluded:

> It is quite likely that history will also record [that] we have been entirely too pessimistic about the possible training potential of the brain injured, and that this pessimism has prevented us from giving them the intellectual and educational stimulation that we would wish for all our children [Gallagher, 1960, p. 168].

Here is evidence that the Strauss-Lehtinen techniques are not necessary to achieve moderately good results with neurologically impaired pupils. In a followup of his subjects to determine the effects of removal of the special tutoring, Gallagher (1960) discovered that the gains were lost in a year after the tutoring ceased. He claimed that the failure of these gains to hold over time may have been a result of the unstimulating environment of the residential facility, and that the gains would likely have been more permanent in a more normal community setting. Finally, he pleaded for an intensive, individualized approach to build conceptualization among young neurologically impaired pupils, rather than almost exclusive devotion or attention to the development of social skills, as has been so often the case when such children are enrolled in special classes for the educable mentally retarded. This position would meet with the support of many leaders in this particular field, including Lehtinen.

Cruickshank, Bentsen, Ratzeburg, and Tannhauser (1961) conducted a two year demonstration study with forty subjects, half of whom were diagnosed as brain injured and half as emotionally disturbed. The brain injured were not only detected by neurological tests, but were also hyperactive and aggressive as well. A typical Lehtinen type classroom environment was created for at least the experimental groups. The investigators concluded that: "While still further evidence needs to be obtained, it is the opinion of the authors that hyperactive children in an unstimulating environment and a structured program demonstrate sufficient progress to warrant continuation of this approach with such children [Cruickshank, et al., p. 421]." In light of the methodological problems encountered and the weakness of the statistical evidence, it is difficult to determine how this statement is justified. One has difficulty in ascertaining whether the subjects were brain injured, emotionally disturbed, hyperactive, or all three. The teachers with contrast groups were allowed to set up any type of treatment they wished, and many of them chose to adopt the Lehtinen techniques similar to those used in the experimental classes. Thus the treatments were confounded. Finally, there is little statistical evidence, in the final report, that the experimental groups made greater progress than the controls; however, if one wishes to accept the conclusions of the authors, we have some support for the use of the Lehtinen procedures with the Strauss type child.

Vance (1956) provided daily, highly structured educational programs in reading readiness over an eight month period for matched groups of non brain injured and neurologically impaired children in a residential school. She found no significant difference between the groups at any time on reading and reading readiness tests, indicating that both groups learned equally well under the treatment she provided.

Frey (1960) conducted a retrospective study which tested the Lehtinen teaching techniques with Strauss type children. He selected a group of 20 neurologically impaired children who, on psychological tests, also exhibited perceptual disorders, and who had been under a special education program using the Lehtinen techniques. These were compared with 20 non brain injured retarded children of similar age and intellect who had been attending conventional programs in regular and special classes. In his survey of the reading behavior of these two groups, Frey found the Strauss type group to be superior in silent reading tests and in sound blending ability. In addition, he found the Strauss type child to have a normal profile of reading areas, while the non brain injured group showed excessive numbers of faulty vowels, faulty consonants, omissions of sounds, and omissions of words. This study has importance because it demonstrates that the Lehtinen techniques appear to work, at least in reading, for Strauss type children; however, the study has the clear weakness of confounding types of subjects with treatments. The non brain injured did not get the special treatment. Whether these techniques will work equally well in other areas of instruction, with cultural familial retardates and even with the intellectually normal, has yet to be demonstrated. Until research is accumulated to the contrary, teachers will apparently be on fairly safe grounds in experimenting with the use of the Lehtinen techniques for teaching children who have the Strauss syndrome. Also, school systems cannot be discouraged from experimenting with special classes for the Strauss type pupil on the basis of the Frey study—but some of the other evidence should lead us to be extremely cautious in advocating the Lehtinen approach uncritically.

Unfortunately, no research evidence was found in the literature on the efficacy of the motor development techniques advocated by the

Doman-Delacato, Kephart, or Schilder-Bender-Friedus groups when applied specifically to children with the Strauss syndrome. However, Robbins (1966) has recently published data on second grade children which suggest that neurological organization is not related to reading achievement, and that the addition of Delacato's motor training program in creeping, walking, etc., to the ongoing curriculum did not enhance reading or lateral development. He concludes that caution should be exercised by anyone considering the adoption of Delacato's theory, since his negative findings cast doubt on its practicability and validity.

Research evidence on the efficacy of perceptual training is also sketchy. However, Forgnone (1966) has shown that Frostig Visual Perception Exercises are effective in increasing scores on the Frostig Developmental Test of Visual Perception for an undifferentiated group of special class educable retardates. Here again, the problem simply is that researchers have not elected to select Strauss type children specifically as subjects for efficacy studies.

Conclusion

In light of the foregoing review of the literature, the following conclusions are suggested:

1. Neurologically impaired children, so identified by physicians, provide such a heterogeneous group of children behaviorally that the diagnosis has little utility for educational treatment, yet most of the studies to date have been done with neurologically impaired children rather than with children who display behavioral characteristics of the Strauss syndrome. Conceivably, the Strauss syndrome is even too much of a catchall. Perhaps, for study purposes, we need to identify subgroups within this classification such as those who are hyperactive versus those with visual perceptual impairments.
2. Except for the Frey retrospective study, there is no empirical evidence to demonstrate the effectiveness of the Lehtinen techniques for teaching concepts, including academic learnings, to the Strauss type child. What is sorely needed is a well designed experimental study to determine whether the Lehtinen techniques are differen-

> tially effective for the Strauss type child as contrasted with the cultural familial retardate, if not the normal child. Another needed study is one which would test the Lehtinen versus the conventional approach in teaching comparable groups of Strauss type children. Thus there is little empirical evidence at this time either to justify, or not justify, special classes for the Strauss type pupil, utilizing Lehtinen techniques. Furthermore, efficacy studies in the areas of motor development and perceptual training with Strauss type children are nonexistent. Research on teaching to weaknesses and behavior shaping utilizing operant techniques have, so far, taken only the form of case studies. Yet educators must teach children in groups; thus investigations involving clusters of pupils are imperative.

My sincerest plea is that behavioral scientists devote greater energy to solid experimentation with differential techniques of teaching children who display the various behavioral characteristics of the Strauss syndrome. It is probable that structured, systematic teaching (applying operant conditioning techniques) will provide us with evidence that Strauss type children can learn much more than we have assumed. Hopefully, in the next decade, research will be forthcoming which will lead us to establish sound educational programs for the Strauss type pupil, based on empirical evidence rather than on theoretical and philosophical positions.

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A Summary of the Literature on Behavior Disorders in Brain Damaged Children

GARY M. CLARK

In his original summary of the literature on brain injury, which covered the period from 1870 to 1945, Klebanoff reported only two studies which investigated psychological problems associated with brain damage in children. Nine years later, Klebanoff, Singer, and Wilensky (1954) compiled another summary of research on the psychological consequences of brain lesions and ablations and this time devoted a separate section to the studies related to child problems. The growth of interest in problems of children during that period stemmed primarily from the Werner and Strauss studies, concepts and investigations of Doll, the writings of Bender, studies by Cruickshank and Dolphin on cerebral palsied children, and single studies by other authors. Since that time, there have been countless studies and publications in this area. This writing is an attempt to summarize some of the developments and trends in the area of psychological disorders in brain injured children since 1954, with special attention being given to diagnostic and educational therapeutic procedures.

In this paper, the term brain damaged, brain injured, or organically impaired will refer to the child described by A. A. Strauss (Strauss and Kephart, 1955):

> A brain-injured child is one who before, during, or after birth has received an injury to or suffered an infection of the brain. As a result of such organic impairment, defects of the neuromuscular system may be present or absent; however, such a child may show disturbances in perception, thinking, and emotional behavior, either separately or in combination. These disturbances can be demonstrated by specific tests. They prevent or impede a normal learning process [p. 32].

The American Psychiatric Association (1952) has used the term brain damaged to associate personality disorders with organic or structural disorders in the brain. Tobis and Lowenthal (1960) report that current concepts tend to support the view that there is no dichotomy between the functional and the organic. The functional designation may represent only the inadequacy of our present diagnostic tools.

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A description of all the symptoms characteristic of brain injury that have been mentioned would entail an enumeration too lengthy for this paper. It is generally agreed that there is no clinical entity with a definite personality pattern displayed in these children. However, there are certain specific characteristics or symptoms which do recur in the educational area (Kaliski, 1955). These include perceptual disturbances, conceptual disorders, and behavior difficulties. Perceptual disturbances occur in the visual, auditory, and kinesthetic fields. Concept formation, reasoning, and comprehension may be fluctuating, incoherent, not integrated, and confused. Tolerance for emotional stress is very limited and once the threshold is exceeded, an emotional state of high tension ensues. Excessive crying which appears to be without cause or out of proportion to the cause is often a characteristic feature of this condition. Involuntary movements may occur as concomitants of excitement or tension.

Beck (1961) has complied a list of symptoms of brain injury indicated by a majority of writers. These include the following:

perseveration
distractibility
disorganization or lack of integration
perceptual difficulties
conceptual difficulties
language disorders
motor incoordination
disparity in development
hyperactivity
emotional instability
irritability
insecurity
mental deficiency
poor retention

Bender (1949) gives a general summary of the condition with which this paper intends to address itself. She holds that psychological problems arise in the organically disturbed child because (a) motor disorders make for prolonged dependency on the mother; (b) perceptual or intellectual problems lead to frustrations, misinterpretations of reality, and bizarre behavior patterns in efforts to make contact with the world; and (c) disturbed patterning of impulses leads to distortions in action patterns with compulsive features. Anxiety, she feels, due to physiologic disorganization but secondary to frustration, is basic to the condition. In one of her more recent writings (Bender, 1956) she reports that modern psychology no longer believes that any symptomatology is the direct result of a defect. The symptom is the expression of the function of those parts of the brain which are preserved.

Recently there has been a movement to deemphasize etiology and classification and direct efforts toward evaluation which delineates areas of strength and weakness among any given child's cognitive motor and personality functions. Advocates of this approach include Kirk and Bateman (1962), Capobianco (1964), Bateman (1964), and Braun, Rubin, Beck, Llorens, Mottley, and Beahl (1965). Clements and Peters (1962) made a case for refinement of terminology in this area and tried to distinguish between minimal brain dysfunction and brain dysfunction which could be considered major enough to indicate mental deficiency. They recommend specific treatment for minimal brain dysfunction in children and seem to reflect the tendency of the medical profession to refine diagnostic procedures, classification, and treatment, rather than accept the functional behavior approach.

Diagnostic Procedures

Establishing the presence of organic disorders is an essential part of the understanding of the brain injured child. Bradley (1955) states that there are five considerations leading to an accurate diagnosis. These include: (a) the distinctive behavior patterns of the brain injured child, (b) his performance on judiciously selected psychologic tests, (c) evidence in the past medical history of a presumptive cause for organic impairment, (d) corroborative evidence of cerebral lesions on neurologic examination, and (e) electroencephalographic evidences of disordered cerebral physiology. Evidence from all five of these areas should establish a diagnosis which is convincing to all. Such complete evidence, however, is rarely available. In many instances, the general behavior pattern, if carefully evaluated, may be sufficient. Evidence from any or all of the other four sources may be considered as corroborative information. Clements and Peters (1962) disagree strongly with this approach and hold that omission of any one of these procedures makes possible a blatant diagnosis.

Questions arising from the problems in diagnosis and proper treatment are legion. Current hypotheses (Graham and Berman, 1961) awaiting testing are: (a) undetected cerebral injury is much more common in children than is usually suspected and may account for a wide range of behavioral deviations; and (b) since the brain of the child is less mature, it is possible that injury to it results in quite different consequences than might occur in an injury to an older person. The former hypothesis was the view of Gesell and the latter is the view of Hebb, who also proposes that there is a problem of generalized and specialized impairment of function. Another current hypothesis comes from Baer (1961) who holds that there is the possibility of schizophrenia being a form of brain damage, a view widely held in medical circles. Most studies concerning diagnosis of brain injury deal with psychological, neuropsychological, and neuropsychiatric tests and examinations which attempt to yield some type of differential diagnosis. The psychological and medically oriented devices which have evolved during the past twelve years will now be reviewed.

Psychological Tests. As we have seen, the symptoms of brain injury can be quite varied, and as they do not necessarily fall into any given pattern, certain implications become apparent with regard to psychological testing. First, there is the problem of determining whether any of these symptoms exist, which means that an extremely large number of abilities and a wide range of behavior need to be sampled and evaluated. This implies that a global approach should be used in the beginning, followed by more specific techniques in those areas suspected to be pathological.

As it is generally held that any insult to the brain will affect the integrity of the entire behavior of the organism, the most general approach to diagnosis usually begins with an assessment of general intelligence. There is an acceptance among clinical workers that level of intelligence in itself is of little diagnostic value in detecting brain injury. Intelligence tests have their value in assessing intelligence and not in the detection of organicity, and should be chosen with care. Some clinicians focus upon the pattern of performance and particular test items in trying to diagnose brain damage as they believe, along with Benton (1961) and Arthur (1958), that brain injured children often show selective impairment in their intellectual functioning. Beck (1956), using a public school group of mentally handicapped children, found that the Wechsler Intelligence Scale for Children (WISC) Performance IQ's of organics were significantly lower than those of nonorganics. In the organic group the performance IQ's tended to be lower than the Verbal IQ's, whereas the nonorganics showed a reverse pattern. He found no characteristic patterning of subtest scores. On the other hand, Newman and Loos (1955), using an institutional population, found that organics show no more difference in WISC Verbal and Performance IQ's than do familial retardates and undifferentiated groups. Haines (1954) did a study using the Merrill-Palmer Scale of Mental Tests and concluded that it was not useful to differentially diagnose brain injured children as separate from problem children or those from foster homes. Berko (1955) found that exogenous mentally handicapped (Strauss syndrome) children had a significantly larger scattering of misses on the Stanford-Binet items than endogenously mentally handicapped children. Rowley (1961), in an analysis of WISC performances of brain damaged and emotionally disturbed children, found a minimum IQ score of 83 and no differences in test patterns. Clements and Peters (1962) report that three patterns of WISC subtests have been isolated in their clinic: (a) WISC Pattern I—scatter in either or both the Verbal and Performance Scales (low scores—relative to the others—most frequently occur in Arithmetic and Digit Span in the Verbal Scale, and Block Design, Object Assembly, Coding, and Maze in the Performance Scale); (b) WISC Pattern II—the Verbal IQ score is 15 to 40 points higher than the Performance IQ score; (c) WISC Pattern III—the Performance IQ score is 10 to 30 points higher than the Verbal IQ score. On the basis of these and other studies, the Stanford-Binet and the WISC have not been found to be significantly valid in the diagnosis of brain damage, but are still used to yield some quantitative measurement. One or more test items in both the Stanford-Binet and the WISC which measure perceptual motor ability may aid significantly in differentiation of organic children, but the diagnostic value of global intelligence or of item or pattern analysis among subtests is still essentially one which must be used advisedly with other information which is obtained.

Simple perceptual functions can be measured and there seems to be conclusive evidence now that the results have diagnostic value (Strauss and Kephart, 1955; Arthur, 1958; Koppitz, 1964). Memory for and reproduction of designs such as those in the Bender-Gestalt test also have been accepted as having diagnostic value (Bender, 1956; Koppitz, 1962, 1964; Clawson, 1962; Guertin, 1954; Holroyd, 1966). In this area, some visual retention tests have been devised by Benton, Graham and Kendall, Ellis, and Bender. Of these, the tests by Ellis and Bender seem to have the most validity. Another type of visual perception test is the Marble Board Test. This is accepted fully by many clinicians but is still lacking in standardization. Studies by Friedman and Barclay (1963) and Ko (1961) suggest cautions in the use of this type of instrument.

Auditory, visual, and tactual figure ground tests have demonstrated that they also have diagnostic value but need more validity and reliability data. Koh's block designs have been found to differentiate organics from nonorganics by the perceptual deficit shown, but the test does not seem to be able to be counted as a completely acceptable instrument yet, as most of the data on its validity has come from studies on mentally defective children.

The Goodenough Draw-a-Man Test continues to be widely used clinically in evaluating children. Bender (1956) has found that a most important diagnostic clue lies in the discrepancies between the Stanford-Binet and Goodenough mental age scores. A Goodenough mental age two years or more below the Binet mental age is considered to be highly suggestive of organic impairment. Use of the Goodenough and similar drawing tests is questioned by Goldenberg (1953) and Clements and Peters (1962).

The Porteus Maze Test was conceived to test one's ability to plan or inhibit and modify behavior in reference to probable consequences of alternative reactions. The common description of impulsivity as a characteristic or symptom of brain damaged individuals suggests that they would do poorly on the Porteus Maze. Studies (Strauss and Kephart, 1955) do show, in fact, that brain injured children do more poorly on this test than those who are not brain injured, but it has been found that presumably noninjured, behavior problem children also have poor performance on this test. More validation is needed to determine the test's validity in differentiating brain injured, behavior problem children from noninjured, behavior problem children.

Various types of sorting tests have been found useful with adult patients suspected of having disorders of thought processes. Some of these tests have been modified and applied to children with brain damage but, in general, they remain of little value as differentiators until adequate schemes for quantifying results are found. These usually give some information on laterality and hand dominance. Doyle (1962) suggests the usefulness of the Harris Test of Lateral Dominance in this area of assessment.

The bulk of clinical investigative work on the capacities of the nondefective brain damaged child has centered on his visuoperceptive and visuomotor performances (Benton, 1962). The approach has been a rewarding one but other possibilities exist which should be thoroughly explored. Graham and Berman (1961) suggest the use of the Differential Language Facility Test and the Hunter-Pascal Concept Formation Test. Beck (1961) reports the findings that the Block Design Rotation Test and the Manual and Finger Dexterity Tests of the General Aptitude Test Battery used by the US Employment Service are consistent differentiators. He also considers the Method or Approach score on the Marble Board Test most promising. Ross (1954) has suggested the tactual perception of form to be a possibility to explore. Frostig (1963) has devised a test of visual perception which includes eye hand coordination, figure ground perception, perception of form constancy, perception of position in space, and spatial relationship.

Studies in progress at the University of Indiana Medical Center by Reed, adapting the work of Reitan with the Halstead Impairment Index for use with young children, appear promising. Reed, Reitan, and Kløve (1965) report that 50 brain damaged children ranging in age from 10 through 14 were matched with 50 normally functioning children on the age variable. Each subject was individually administered a modification by Reitan of the Halstead battery of neuropsychological tests. The battery included the Category Test, Halstead Speech Sounds Perception Test, Halstead Tactual Performance Test, WISC, Finger Oscillation Test, Time Sense Test, and Trail Making Test. The brain damaged subjects performed significantly less well than the control children on all of the tests, with differences between the groups occurring more frequently on the tests of language functioning than on any other test. Reed and Fitzhugh (1966) reported similar results in another study in which deficit patterns consisted of relatively greater impairment on tests of language and symbolic ability as compared with tests of immediate adaptive ability.

Benton (1962) suggests that the areas of linguistic behavior, reasoning, or such more or less general characteristics as "behavioral flexibility" have yet to be explored to any extent. Barsch (1962) and his associates have devised a clinical tool for their own use in evaluating the state of a child and his capability for meeting daily demands and for learning. It is called a Functional Organization Scale. It yields a composite assessment of the child including normative scores, observative behavior, and deductive inferences. This type of descriptive tool suggests the degree of behavioral flexibility to which Benton was referring.

The emphasis on early diagnosis has resulted in some efforts in development of preschool instruments. Graham, Ernhart, Craft, and Berman (1963) developed measures of vocabulary skill, conceptual ability, perceptual motor ability, and personality characteristics of preschool aged children. Particular procedures were selected either because they had successfully differentiated brain injured from normal adults or because they measured functions relevant to theoretical questions concerning the brain injured child. Using these measures, Ernhart, Graham, Eichman, Marshall, and Thurston (1963) compared the performance of normal preschool children with 70 brain injured children. The brain injured children were significantly, but not equally, impaired in all areas measured. Personality functioning was significantly less affected than nonpersonality functioning. Neither the hyperkinetic personality syndrome nor the differential pattern of impairment seen in adults was found in their heterogeneous sample of brain injured children. It was suggested that there are systematic differences in the effects of injury, depending upon the age at the time of the injury.

There have been numerous studies describing single instruments which differentiate brain injured populations from non brain injured populations. Some of these may prove to be valid enough for inclusion in a battery of psychological and/or neuropsychological instruments. Some of these include the perception of autokinetic movement (Bennett and Poit, 1963); the Figure Square test (Jirasek, 1962); the Organic Integrity Test (Tien and Williams, 1965); a weight discrimination task (Jenkins, 1965); and the Oseretsky-Gollnitz psychomotor test, objectively scored graphometrics, and a ring stacking test (Regel, Parnitzke, and Fischel, 1965).

Medical Tests. Traditionally, in evaluating the brain damaged individual, a thorough neurological examination has often been the sole prerequisite for diagnosis and treatment. Today, however, experience in the fields of physical medicine and rehabilitation have demonstrated that the basis for an effective evaluation must be the addition of a rehabilitative evaluation of the individual's functional capacity and a psychological assessment. The rehabilitative evaluation is basically an evaluation of the neuromuscular functions. Therefore, an evaluation of the brain damaged patient today should consist of the following areas of study (Tobis and Lowenthal, 1960): (a) a neurological examination; (b) a measure of neuromuscular function such as walking, manual dexterity, finger dexterity, etc.; and (c) an evaluation of the psychological and emotional capacities. The psychological evaluation has been discussed previously so the emphasis in the remainder of this section will be on the medical approach.

Those areas of the central nervous system in which a deficit may lead to significant changes in behavior are the sensory sphere and the motor sphere. The general and special sensory structures interpret the external and internal environment of the child, and damage to this area generally disturbs the sensory input at the integrative level. Motor function is essentially the end result of sensory input, and thus a deficit in this area can be effected by insult to the sensory or the motor spheres. The neurologist evaluates the child with special emphasis on history and symptomatology in these areas. Laufer, Denhoff, and Solomons (1957) found that the use of Gastant's photometrazol technique is useful. This is a clinical neurophysiological test which provides a method for the exploration of certain substructures such as the diencephalon and thalamus.

Use of the electroencephalogram, or EEG, as a diagnostic tool has been urged by many. Taterka and Katz (1955), Weir and Anderson (1958), Kennard (1959), Schwade and Geiger (1956), Clements and Peters (1962), Knott (1960), and Winfield (1961) are unanimous in their findings as to the effectiveness of the EEG as a supplementary tool to neurological and psychological information. Of 181 school children with severe school adjustment problems, 75 percent had abnormal EEG's, according to Weir and Anderson (1958). Winfield (1961) states that a majority of the patients referred to him for acting out behavior, convulsion, or headaches show the 6 and 14 per second positive spike pattern and require a persistent regime of medication. He further reports that although an EEG may not reveal any abnormal pattern when organicity does exist, an EEG which does show an abnormal pattern is generally diagnostic. Kennard (1959) conducted a study comparing disorders in thinking with findings on the EEG. Her results were that in the younger age group, organic brain injury and autistic or schizoid thinking patterns are strongly related to data from social and developmental case histories. This was supported by a study by Hughes (1965).

Virtually no published work was found in the literature relating impairment of function to localization in children. Barsch (1962) reports that investigations in this area are in progress at Northwestern University and Indiana University Medical Center. The most comprehensive research project to date in the diagnosis and etiology of neurological disorders is now in progress through the joint efforts of 14 medical centers and the National Institute of Neurological Diseases and Blindness. The Collaborative Perinatal Research Project was begun in 1959 and will include 60,000 women and the children born to them. Followup will continue until the children's twelfth year of age.

Educational Therapeutic Procedures

Ultimately the problem of the brain injured child with behavior disorders is a problem of learning. He needs to learn new response patterns which are acceptable and extinguish old response patterns. Learning can come through formal education, response conditioning, therapy, or combinations of each. Learning by any of these processes is thought to be enhanced by certain medications in many cases. A further discussion of these approaches will now follow.

The basic assumption of many professionals in this field is that a formal, systematic educational approach, different from the conventional approach, is needed for the disturbed brain injured child. The emphases for these educational provisions have included Lehtinen's (Strauss and Lehtinen, 1947) restricted environment, Kephart's (Kephart, 1960) perceptual motor techniques, Doman and Delacato's (Doman, Spitz, Zucman, Delacato, and Doman, 1960) neurological patterning, Frostig's (Frostig, 1963; Frostig and Horne, 1964) visual perceptual procedures, Epps' (Epps, McCammon, and Simmons, 1958) visual-auditory-kinesthetic-tactual perceptual techniques, Kirk and Bateman's (1962) tutoring techniques for specific learning disabilities, and the eclectic approach of Friedus (1964).

Daly (1965) has reviewed the research in the area of educational provisions and reports an obvious lack of formal experimentation to support the general assumption that mild brain damage, regardless of intellectual level, requires a different mode of education in the schools than the non brain damaged child. While this still remains the prevailing assumption by some educators (and many parents), it has been seriously questioned by a number of researchers. Conventional curriculum approaches for the brain injured and non brain injured seem to be equally effective as Capobianco (1954, 1958) and Bensberg (1952) found no significant differences between these two groups in reading and arithmetic skills. Special curriculum approaches have yielded similar results. Cruickshank, Bentzen, Ratzeburg, and Tannhauser (1961) reported that while significant visual perceptual gains after one year of special training were indicated, a followup one year later revealed a loss in these gains. By the end of the second year both the experimental and the control groups revealed increases in achievement with the overall differences being nonsignificant. Kirk (1958) found that under early educational stimulation the rate of growth of organic mental retardates did not increase as rapidly as the nonorganic retardates under the same stimulation program, although some of the organics did demonstrate significant growth. Gallagher (1960, 1962) did obtain positive results with institutionalized brain injured retardates through tutoring procedures but gains were dissipated after training was terminated. Burnette (1961) and Vance (1956) also found no significant gains with brain injured children using special educational techniques. Frey's study (1960), on the other hand, is the only study found which supported special techniques by Lehtinen and this study was limited to the teaching of reading.

Robbins (1965) reports that his review of the literature in the area of special educational and training efforts based on neurological organization revealed that, while the popular media have generally been favorable toward such a theoretical position, writers from the areas of medicine, psychology, and education have not shared this interest and enthusiasm. He reported that he was unable to discover any published research reports other than their own writings (Doman, et al., 1960) which support the theory. Robbins' own study and those of Dunsing (1966) and Evans, Ritter, and Hall (1966) were negative in their findings through attempts to correlate this theory, and its proposed techniques, with empirical evidence. Robbins suggests that the lack of research support would indicate that verifiable, replicable, empirical evidence from controlled studies using acceptable research methods is needed if the proponents of this theory wish to gain professional acceptance and recognition from the scientific fields.

The Epps (Epps, et al., 1958) and Frostig (Frostig, 1963; Frostig and Horne, 1964) techniques are also awaiting general validation through acceptable research methods. The Gallagher (1960) and Kirk and Bateman (1962) studies are examples of how controlled experiments and documented case studies can add to the body of knowledge in this area. Whether the research is done to validate one approach or theoretical orientation over another, or whether it tries to substantiate the validity of remediating to weakness or strengths is not the issue. The issue is that research is needed in which certain educational or remediation procedures would be used with one group, alternative or diametrically opposed procedures used with a second group, and control techniques or no treatment with a third. Results from such studies would shed some positive light on the whole issue of educational methodology.

Efforts to develop special classes in average school settings for the brain damaged child have been few. In a practical way, all schools have to cope with the emotionally disturbed, whether it is known that the reason for the disturbance is organic or not. Disturbed pupils are excluded from school, demoted, promoted, or just permitted to flounder. In the past, these children have been considered almost completely outside the areas of school responsibility. But during the past twelve years the attitude has rapidly begun to change, giving rise to experimental programs for dealing with disturbed brain injured children. Some of these programs will be described below.

Joliet, Illinois. The first major experimental program was at Joliet, Illinois (Jolles, 1956). The researchers there followed the Strauss and Lehtinen procedures regarding the removal of all distracting stimuli in the environment; a quiet, steady routine; small class; isolation of individuals from the group so that attention can be focused on the materials for study, and the simplification of presentation of all perceptual motor skills. The first published report presented the following conclusions which have served to encourage the initiation of similar programs:

1. Children in special class made more progress than is usually made in classes for educable mentally retarded, where most brain damaged children are placed.
2. Special classes for brain injured children can be adapted to a public school situation.
3. The school psychologist can contribute to the establishment of such a class by screening and selection and through community education.

4. Selection of children should be done cooperatively by the school psychologist and a neurologist.
5. The teacher must have a wide background of experience and it is desirable that she have two years of training in the education of the brain damaged.
6. Three years should be the maximum time spent by any child in the classroom.
7. School districts contemplating establishment of classes should have classes for educable mentally retarded already established so that children can have some transitional placement before placement in a regular classroom.

Arlington, Virginia. Kaliski (1959) reports that a structured environment is the key to the education of the organically impaired because of the nature of the condition. She feels that because of the physical impairment to the brain, the world around the child may be perceived in a diffused, chaotic, structureless conglomeration of visual, auditory, and kinesthetic impressions and the child needs help in bringing order into that chaos. The Arlington program emphasizes this type of structure by providing an environment with clear direction, firm expectations, and consistent followup.

Denver, Colorado. McCartney (1954) reports a program in Denver built around audiovisual, tactual, and kinesthetic materials, but involving no projects or activities, since these tend to provide too much distraction.

Houston, Texas. Hemmer (1964) reports the progress being made in a new class for the organically impaired which has a structured program and a structured environment, and pays much attention to the elimination of distracting stimuli.

Montgomery County, Maryland. Cruickshank, et al. (1961), and Tannhauser (1964) report on the pilot study conducted in Montgomery County which was developed to investigate the value and effect of a nonstimulating classroom environment, specially prepared teaching materials, and highly structured teaching methods upon the learning problems and school adjustment of hyperactive, emotionally disturbed children with and without clinically diagnosed brain injury. The plan of education involved Cruickshank's modification of the Strauss-Lehtinen concept of education and was interpreted as showing significant educational and adjustment gains over the control group, but these interpretations are subject to criticism.

Dixon, Illinois. Gallagher (1960) conducted an experimental study in an institutional setting at the Dixon State School for the Mentally Retarded. The purpose of his study was to determine if individual clinical tutoring could specifically improve the functioning of brain injured mentally retarded children in the areas of verbal and non-verbal intelligence, language development, perceptual abilities, quantitative thinking, personality development, and social maturity. The tutoring method was based upon an individual approach which emphasized motivation for learning, success experiences, and utilized the game approach to all learning tasks. The results of his study were summarized as follows:

1. Improvement in the intellectual development of some brain injured mentally retarded children can be obtained through the tutoring methods described.
2. The children who responded to the tutoring achieved more in the area of verbal skills than non-verbal skills, but all of the children had extreme difficulty at the higher abstract levels of conceptualization.
3. The younger children (ages eight-ten) in the study showed significant improvement over the older children (ages ten-twelve).
4. Certain behavioral changes were noticed during tutoring, principally an increased ability to pay attention.
5. When the tutoring procedures were removed from the life of the child there was a tendency for his development to regress to lower levels or become arrested.
6. There was an impressive range of individual differences both in the characteristics of the children prior to tutoring and in their response to tutoring [p. 151].

Implications for educators and therapists from this study would be directed toward the possibilities of utilizing this approach with brain injured children with average or above average intelligence.

These few examples of experimental or established programs illustrate the types of approaches generally being used. The concepts of Strauss, Lehtinen, and Kephart are still the major guidelines for the procedures and practices of school programs today, in spite of the lack of empirical evidence to support their use.

Rachman (1962) has given a succinct overview indicating what the application of learning theory can mean to the education and/or therapy of exceptional children. He recognizes that advances in the application of learning theory to clinical problems in adults have not been accompanied by similar advances in child psychology. He holds that the nature of therapy for these two groups is probably the primary factor. Behavior therapy has so far provided more techniques for the elimination of unadaptive behavior than for the development of desirable behavior. The disturbances of behavior in children are more often of the deficit type and require the building up of adequate behavior patterns.

Operant Conditioning. The recent deluge of operant conditioning procedures into clinical psychology seems to provide a tool for developing deficient responses in children. Lindsley (1956, 1960) followed up the proposals of Skinner (1959) and Skinner, Solomon, and Lindsley (1954) and has produced some interesting analyses of the behavior of psychotic adults. The clinical applications of this technique in child psychology is evident in the work of Ferster (1961), Ferster and DeMeyer (1962), Lovaas (1961), Spradlin (1961), and Bijou (1961) among others. It has been demonstrated that operant conditioning methods can be used to generate and/or sustain stable behavior patterns. Other advantages of operant methods are that they permit, when required (a) nonverbal operations, (b) strict control of variables, (c) quantification of operations, (d) exclusion of clinician variables, and (e) single case studies. Disadvantages are of a practical nature. Operant methods usually demand special equipment and experimental rooms and can be time consuming.

Few studies on operant methods are specifically related to behavior disorders in brain damaged children. Researchers in this field and advocates of behavior modification techniques can explain this, however, by claiming that a basic assumption of this approach is that etiology and classification terminology are not important. They are concerned only with functional behavior. Whelan and Haring (1966) have critiqued this methodology, and state:

> Whether these techniques achieve satisfactory results when compared to systems which have different behavioral approaches can only be resolved through systematic research. Data thus far reported from laboratory experiments and a few studies with small groups of children have demonstrated a high degree of reliability. These data have yet to be validated in regular and special classroom situations. Skeptical, cautious acceptance and application of behavioral modification techniques are certainly indicated [p. 288].

Specialized Therapy. Therapy for the brain injured child with emotional disturbance is beginning to be more specialized with recognition of the unique characteristics of the condition. Bender (Frampton and Gall, 1956) gives some insight into the dynamics of the means by which organic impairment can result in emotional disturbance in pointing out the lack of integrating ability in perceptual experiences. She views this specific disability in integrative function as a clue to the frustration which the child with organic brain impairment suffers. His constant show of energy and drive to make contact with the world and the resulting lack of satisfaction lead him to increase the drive to make meaningful contacts. The continued cycle is depicted in his hyperkinesis or drive to see, hear, feel, touch, grasp, and finally incorporate and destroy every object that cannot be experienced satisfactorily otherwise.

Leland and Smith (1962) and Lawrence (1960) indicate that the primary goals for these children are (a) recognizing self, (b) understanding that impulses can be controlled, and (c) learning to live within social boundaries. As opposed to the structured environment approach in a school setting or the semistructured play therapy setting, Leland and Smith hold to the concept of unstructured materials and unstructured therapeutic goals as being most suitable for these children. This is based on the philosophy that the children have great difficulty in dealing with abstract concepts and in utilizing factors which are usually based upon imagination. The unstructured materials do not set up preconceived notions as to their use, as toys do, but rather promote original thinking. The rewards children receive for demonstrating any original thinking or imaginative ideas help to carry over into the problem areas. In summary, Leland and Smith (1962) feel the whole therapy process is a conditioning process which involves (a) conditioning the child to the idea that his behavior and his ideas are his own and that he is responsible for them; and (b) conditioning the child to organize his behavior around mutual or socially cognitive associations. The whole process forces the child to think, which traditional play therapy may or may not do.

Kirk (Kirk and Weiner, 1963) holds the view that psychotherapy as a single influence on the adjustment of children may not prove to be the most effective approach to modify the behavior of children. He raises the possibility that milieu therapy or the so called non depth therapy with parents and teachers as a part of the therapeutic team may be the most practical and also the most effective method. Doris and Solnit (1963) support this position and state that these children can be impressively responsive to broad provisions within the community.

The combination of therapy and educational provisions is the most recent concept in the treatment of the organically impaired child. This can be accomplished in residential treatment centers, special classes, or by the individual tutorial therapeutic approach. Kaliski (1955) recommends a therapeutic approach which sets objectives but no standards, which allows the child to progress at his own pace, which uses audiovisual and tactual kinesthetic techniques, and which provides guidance for the child's family. Kurlander and Colodny (1965) also advocate a combination of therapeutic and educational procedures.

Rubin and Simson (1960) list child guidance clinics, residential treatment centers, hospital units, and day care centers as current resources for severely disturbed children. The residential treatment center has been the most rapidly advancing resource, but many are privately owned and fees are beyond the means of the average family. Hospital units are generally utilized for inpatient diagnostic study or relatively short term intensive treatment. Day care centers are designed to render first aid or to dilute some of the problems engendered by residential care, and at the same time to provide direct therapeutic help to the child. It has the advantage of not bringing about a total separation from home and community.

All of the settings which have been described above emphasize the integration of clinical service with educational and social programing directed to the treatment of the child in relation to his emotional disturbance. The contribution beyond the child guidance approach is felt to lie in the increased cooperation of several disciplines oriented toward a consistent, total treatment program. However, there are strong indications that a setting has significant therapeutic value when it involves a special educational program that provides an atmosphere adjusted to the child's readiness to gain satisfactions from learning. Rubin and Simson (1960) propose that a logical extension of current facilities is a clinically oriented day school program whose primary aims are accomplished through educational methods. Early identification of disturbances and provision of prophylactic care through special educational methods and clinical understanding would be the primary goals of a program of this kind. Programs of this kind are in the early stages of existence and little has been reported in the literature as yet.

Drugs. Drugs are being used increasingly in an attempt to help stabilize the child's emotions and hyperactivity and make him more amenable to psychotherapy or educational treatment. Levy (1959) and Walker and Katz (1958) support the use of drugs for the improvement of the children and for the relief of pressure and anxiety in parents as a result of the children's improvement. Knobel (Knobel, Wolman, and Mason, 1959) reports that Ritalin has some usefulness; Laufer and Denhoff (1957), Gross and Wilson (1964), and Clements and Peters (1962) advocate selective use of amphetamines; Fish (1960) reports that the phenothiazines, and sometimes Benadryl, reduce psychomotor excitement and indiscriminate response to stimuli. Gross and Wilson (1964) report the use of anticonvulsants on 45 children and state that one-half improved significantly and ten improved dramatically. Methsuximide (Celontin), acetazolamide (Diamox), and d-amphetamine (Dexedrine) were found particularly useful. Clements and Peters (1962) found that the drugs most helpful in their clinic in reducing hyperactivity and irritability and in increasing attention span were captodiamine hydrochloride (Mellaril) and the amphetamines. Eisenberg (1964) has done extensive research in this area and has written numerous articles on drug therapy. He indicates that drugs can be useful in managing pediatric psychiatric disorders when chosen appropriately and applied with discrimination. He states that when a phenothyazine is indicated in the treatment of a major psychiatric disturbance, the drug to be preferred with children is chlorpromazine, because of its wider margin of safety for extrapyramidal manifestations. Diphenylmethane derivatives are mild tranquilizers which have been used to treat behavior disorders, anxiety neuroses, and hyperkinetic syndromes. Stimulants such as dextroamphetamine and methylphenidate have their greatest usefulness in the treatment of the overactive and distractible child.

Summary

Clinical studies of brain injured children with behavior disorders consistently reveal the presence of (a) variability of emotional behavior, (b) sensorimotor disturbances, and (c) a variety of other problems, including learning and adjustment problems at school. Diagnostic plans which are typical of the more recent approaches in differentiating between organically and nonorganically impaired children include (a) careful case histories, (b) neurological examinations, (c) batteries of psychological and personality assessments, and (d) electroencephalograms. Treatments for the condition of organic impairment include special educational procedures, psychotherapy, operant methods, tranquilizing medications, and parent guidance and counseling. These services are provided at facilities such as public schools, child guidance clinics, residential treatment centers, hospital units, and day care centers.

The developments of the past twelve years, as described in the literature, have primarily been that of improvement and refinement, rather than of innovation. The most progress has been shown in the diagnostic tools used in this area.

The prognosis for children with organic problems depends on the way in which they are accepted and assisted, rather than upon the presence or degree of the organic factors themselves (Bender, 1949). The factors which are not related to the organic impairment itself are those factors which must be concentrated upon so that the abilities are emphasized, rather than the disabilities.

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Neurological Organization: An Evaluative Review of the Theories and Procedures of Doman and Delacato

CHARLES W. McDONALD

Twenty years ago in Philadelphia a team consisting of a brain surgeon, a physiatrist (an M.D. specializing in physical medicine and rehabilitation), a physical therapist, a speech therapist, a psychologist, an educator, and a nurse, each discouraged by the ineffectiveness of their several attempts to help rehabilitate neurologically impaired persons, formed an interdisciplinary team.

One of their first tasks was to identify the group with whom they would work. According to Doman (1963) there were three types of children who were often mistakenly classified together:

> The three kinds of children who were constantly put together were deficient children with brains which were qualitatively and quantitatively inferior, psychotic children with physically normal brains but unsound minds, and finally truly brain injured children who [had] had good brains but which had been physically hurt [p. XI].

The team focused the whole of its attention on this latter group of children who had suffered injuries to a brain which at conception was presumed to be perfectly good.

Being discouraged by the results of the symptom centered treatment procedures current at that time, they concluded that if they were to solve the problems created by the multiple symptoms of the brain injured child they would have to attack the source of the problem and approach the human brain itself.

> We held the simple belief that to treat the symptoms of an illness or injury, and to expect the disease to disappear, was unmedical, unscientific and irrational, and if all these reasons were not enough to make us abandon such an attack, then the simple fact remained that brain injured children approached in such a manner never got well. On the contrary, we felt that if we could attack the problem itself, the symptoms would disappear spontaneously to the exact extent of our success in dealing with the injury in the brain itself [Doman, 1963, pp. 11-12].

Once having made this commitment to central rather than peripheral treatment they began an extensive study, not of physiological and/or neurophysiological growth and function, but of the developing behavioral patterns of children. They took theoretical concepts from the realm of functional neurology and, using them as models, related them to the data of developmental psychology. Again quoting Doman:

> First we tackled the problem from a nonsurgical standpoint. In the years that followed, we became persuaded that if we could hope to succeed with the hurt brain itself, we would have to find ways to reproduce in some manner the neurological growth patterns of a well child. This meant understanding how a well child's brain begins, grows and matures. We studied intently many hundreds of well newborn babies, infants and children. We studied them very carefully.
>
> As we learned what normal brain growth is and means we began to find that the simple and long-known basic activities of well children, such as crawling and creeping, are of the greatest possible importance to the brain. We learned that if such activities are denied well children, because of cultural, environmental or social factors, their potential is severely limited. The potential of brain injured children is even more affected [Doman, 1963, p. XII].

Not all of their work was or is behavioral in nature. Neurological and neurosurgical examinations have always constituted an important part of the team's overall effort. Every child seen is subject to an exhaustive neurological examination and, when necessary, neurosurgical examination and treatment are undertaken. The brain surgeons associated with the team have been in the forefront of the development of such procedures as the V-J Shunt for hydrocephalus and the hemispherectomy for traumatically injured children. In addition to this, they experimented with many other new and adventurous methods for improving the total functioning of persons with central nervous system injury.

As the work of the group has grown, they have found it necessary to divide their functions and to develop a series of institutes. There are six institutes at the present time. Four of the institutes are directly involved in the treatment of patients (Institutes for the Achievement of Human Potential, 1965).

The Children's Evaluation Institute has as its function the complete neurological evaluation of all possibly brain injured children who are referred for such evaluation. It uses all available previous testing as well as its own testing procedures to determine if the child is truly brain injured and if he is a candidate for treatment.

The Institute for Neurological Organization undertakes the outpatient treatment of brain injured children who have been accepted for such treatment. It prescribes the program of treatment for the patient and reevaluates him at approximately two month intervals, making appropriate changes in the patient's program in accordance with his progress.

The Rehabilitation Center at Philadelphia undertakes the inpatient treatment of patients who are immediately postoperative or whose problems are of so severe a character that they cannot initially be dealt with at home.

The Institute of Reading Disability diagnoses and treats reading problems in children. Such children are reevaluated approximately every three months and appropriate changes are made in the treatment programs which are pursued in the children's homes.

The other two institutes do not deal directly with patients. The Teaching Institute instructs professional workers in the concepts and procedures of the institutes. Students in the Teaching Institute include physicians, educators, therapists, and professional persons from the allied life sciences and humanities. The function of the Research Institute includes clinical and statistical research in the field of brain injury. Statistical studies are made of the results obtained by means of the procedures employed by the sister institutes engaged in the evaluation and treatment of patients. Clinical studies are directed toward the better understanding of cerebral function and its impairment by brain injury.

Levels of Neurological Organization

The biogenetic law which states that ontogeny recapitulates phylogeny is accepted by Doman and Delacato. They claim that the progression of neurological organization proceeds vertically to the cortex as myelenization takes place. According to Delacato:

> These progressive organizational stages are chronologically predictable. The orderly and sequential myelenization and organization of the sub-cortical areas is pre-requisite to the subsequent proper organization at the level of cortex. They are both pre-requisite to the establishment of complete dominance [1963, p. 47].

This progression begins during gestation and is normally complete by eight years of age (Delacato, 1963).

1. *Cord and Medulla.* During gestation and up to the time of birth the spinal cord and medulla oblongata are the upper reaches of neurological organization. Here lie the ancient and primitive reflexes whose basic contribution to neurological organization are muscle tone, reflex movement, and the preservation of life. The medullary functions continue to be of primary importance at the time of birth since they control by reflex such vital life preserving functions as cardiovascular activity, gastrointestinal activity, and breathing reflexes. At this level, the infant's mobility is undulating and fish like in character. As the newborn makes the transition from a fluid to a gaseous environment, proper medullary function is vital to survival. The infant at this level has movement but no mobility. His movements consist of crude trunkal movements not oriented toward any objectives. It is a totally reflex synergy. The infant lives at this level until about sixteen weeks of age, at which time he leaves his fish like existence and moves on to the next level of neurological organization.
2. *Pons.* The next higher level is the one which is similar to amphibians and is the responsibility of the pons. The pons is the physiological seat of the tonic neck reflex. Ontogenetically this reflex should be partially established prior to birth; its reflex function tends to cease at about twenty weeks of age. The first use of the tonic neck reflex takes place *in utero.* The mere turning of the head flexes the arm and legs in the direction in which the head is turned. The tonic neck reflex allows the foetus its higher level of serialized movement. An intact tonic neck reflex pattern is prerequisite to nontraumatic or normal birth, the optimal posturing and function-

ing of the body for crawling, and ultimately for organized seeing and hearing. The establishment of the tonic neck reflex pattern permits the homolateral pattern of crawling and the establishment of biocular vision and biaural hearing. The organization at the level of the pons is typified by the first functional movement, the beginning of true auditory and visual reception, and generally increased movement.

3. *Midbrain.* At about six months of age the infant begins to move into the midbrain stage of development. At the midbrain level we begin to see other relationships to mobility; for the first time the third dimensional movement in the form of creeping appears. This requires audition binaurally, vision binocularly, and the direct relationships of visual fibers, auditory fibers, labyrinth, occulomotor nerves, light and posture reflexes, and the muscles of the neck and trunk, in forming the level of behavior indicated by myelenization and organization at the midbrain level. The child also learns to creep in cross patterning rather than in homolateral patterning.

   At the midbrain level the child becomes a truly land animal. We could wonder if the child is destined to be a quadruped or a biped when observing him at the midbrain level. This phase of neurological organization is in reality a preparatory phase for making the infant ready to assume his human upright position when he achieved all of the functions of the midbrain level.

4. *Cerebral Cortex.* At about one year of age the child moves from a midbrain type of overall function to the level of *early* cortical function.

   The child at this level is becoming increasingly proficient at bilateral activity. He shows much improvement in bilateral control, then begins to experiment with becoming paralateral. He begins to use his hands and arms independently of his feet and legs and masters one of his most human functions, that of walking. This higher level of neurological organization permits not only walking but also the development of stereophonic audition, stereoscopic vision, and stereognostic touch. The child also learns to speak and acquires language.

From 12 to 18 months of age the child operates at an early cortical level. The progression of neurological organization now becomes painfully slow, when compared with the explosive growth which took place from birth to one year of age. He needs seven more years to develop from early cortical function to the completion of neurological organization. [*Editor's Note:* The author is apparently not saying here that *neural growth* is complete at eight years, since it is commonly believed that structural maturity of the central nervous system is reached sometime between age 12 and age 16. He is specifically referring to the construct, *neurological organization.*]

The completion of this stage of neurological organization places the child somewhat ahead of the primate. He is now in the stage of *later* cerebral cortex functioning. He can now walk bilaterally in a cross pattern—that is, by swinging the opposite arm toward the forward leg on one step, and reversing these positions on the next step. He now has stereopsis and stereophonic hearing, and has developed stereognosis. He has mastered enough paralateral activity at the cortical level so that he can now oppose his finger and thumb dexterously and, as a result, has fairly sophisticated bilateral manual dexterity and can supinate and pronate his hand and forearm. These achievements of the young child help him to move about his environment developing greater muscle strength and greater vital capacity and gaining experiences upon which to build later intellectual function. These are the final vertical achievements possible in neurological organization.

Now, after a period of a few years, the child must move on to the next stage of neurological organization—that of *laterality*—a stage which is unique to man. He has already become a truly expressive organism with a spoken language. He must now proceed to the stage of a more sophisticated language function, one which includes reading, writing, and spelling.

The next step in the progression toward complete human function is the development of cortical hemispheric dominance. Up to this point in cortical functioning, both hemispheres of the cortex

> operate in concert within a balanced transcortical relationship. At the laterality stage, the two hemispheres begin to develop differentiated functions—one becoming dominant and the other assuming a subdominant role.
>
> Brain injury or inadequate subcortical organization both result in difficulty in the establishment of hemispheric dominance. This final lateral development takes place between five and eight years of age, i.e., after this age, lateral dominance is rigidly resistant to change.

Delacato (1933) claims:

> We can corroborate the progress of neurological organization clinically. The mobility functions of growing and maturing children indicate the level of neurological organization they have reached. There are many other indices. They become less hyperactive as they grow older, they learn with greater facility, indeed they generally follow the patterns outlined by Gesell in all of his studies. These changes in behavior, which Gesell put into the literature and to which many others have added, now make up the body of knowledge known as child development. There is complete agreement that these changes are the result of changes which take place within the child [p. 65].

If we juxtapose the phylogenetic development of the nervous system and its significant functions with the ontogenetic development of a single human being who is on his way toward neurological organization resulting in speech and reading, we find some interesting relationships. These are presented in Table 1.

Using this model of neurological organization and behavioral criteria generated from it, Doman and Delacato are able to diagnose and prescribe treatment for brain injured children.

Diagnostic Criteria

The criteria developed by Doman and Delacato measure brain function in six areas in which man has attained capabilities beyond those of any other living organism. The three expressive or motor functions include mobility, language, and manual competence (writing). The three receptive or sensory functions are visual competence (reading), auditory competence (understanding man's spoken language), and tactile

TABLE 1

Integration of Phylogeny and Ontogeny in the Process of Functional Neural Growth

	Highest Neurological level	*Mobility*	*Vision*	*Audition*
Newborn Infant	Medulla	Trunkal Movement	Reflex	Reflex
Fish	Medulla	Trunkal Movement	Reflex	Reflex
Four month old Infant	Pons	Homolateral Crawling	Biocular	Binaural
Amphibian	Pons	Homolateral Crawling	Biocular	Binaural
Ten month old Infant	Midbrain	Cross pattern Creeping	Binocular Yoking	Binaural
Reptile	Midbrain	Cross pattern Creeping	Binocular Yoking	Binaural
One year old Infant	Early Cortex	Crude Walking	Early Fusion	Early Stereophonic
Primate	Early Cortex	Crude Walking	Early Fusion	Early Stereophonic
Eight year old (who speaks, reads, and writes)	Cortical Hemispheric Dominance	Cross pattern Walking	Stereopsis with Predominant Eye	Stereophonic Hearing with Predominant Ear

competence (stereognosis). These areas and their functions are presented in Table 2.

The rates of development differ among the various functions. Since the development of the sensory tracts precedes that of the corresponding motor tracts, it is commonly found at the institutes that the performance level is higher on the receptive dimension than on the motor dimension. At the initial evaluation, the level of each of the six functions is recorded on a chart which provides a developmental profile. This not only gives an initial base line which expresses the Neurological Age of the patient when he is first evaluated, but also permits ready comparison of the functional response of the individual to treatment at subsequent evaluations. (See Figure 1)

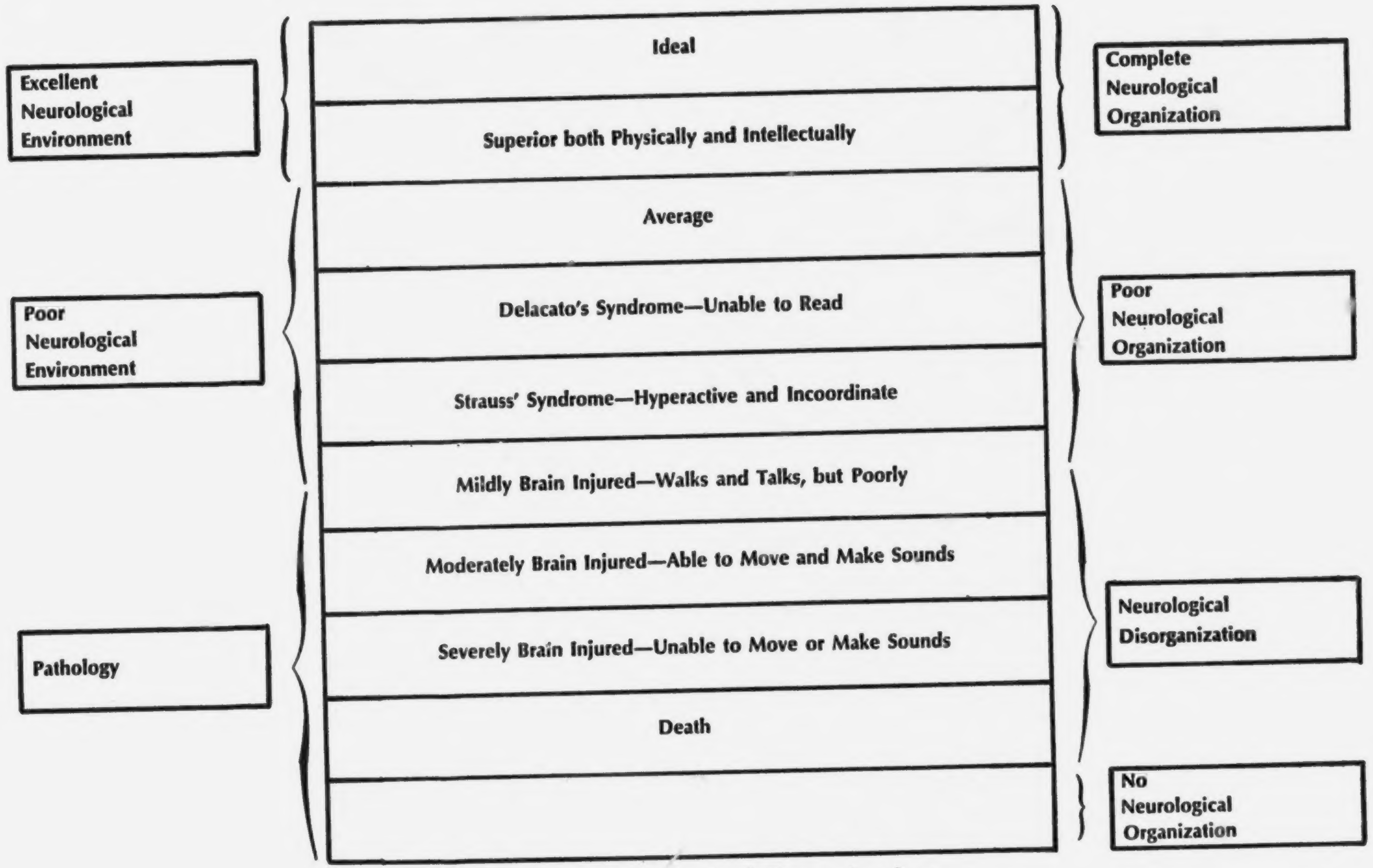
Excellent Neurological Environment
Poor Neurological Environment
Pathology
Ideal
Superior both Physically and Intellectually
Average
Delacato's Syndrome—Unable to Read
Strauss' Syndrome—Hyperactive and Incoordinate
Mildly Brain Injured—Walks and Talks, but Poorly
Moderately Brain Injured—Able to Move and Make Sounds
Severely Brain Injured—Unable to Move or Make Sounds
Death
Complete Neurological Organization
Poor Neurological Organization
Neurological Disorganization
No Neurological Organization

FIGURE 1. *Neurological Organization*

TABLE 2 *Development Profile*

			Expressive or Motor	
Brain Stage		*Time Frame*	*Mobility*	*Language*
VII	CORTEX	Superior 36 Months Average 72 Months Slow 96 Months	Using a leg in a skilled role which is consistent with the dominant hemisphere	Complete vocabulary and proper sentence structure
VI		Superior 22 Months Average 46 Months Slow 67 Months	Walking and running in complete cross pattern	2000 words of language and short sentences
V		Superior 13 Months Average 28 Months Slow 45 Months	Walking with arms freed from the balance role	10 to 25 words of language and two word couplets
IV		Superior 8 Months Average 16 Months Slow 26 Months	Walking with arms used in a primary balance role most frequently at or above shoulder height	Two words of speech used spontaneously and meaningfully
III	MID BRAIN	Superior 4 Months Average 8 Months Slow 13 Months	Creeping on hands and knees culminating in cross pattern creeping	Creation of meaningful sound
II	PONS	Superior 1 Month Average 2.5 Months Slow 4.5 Months	Crawling in the prone position culminating in cross pattern crawling	Vital crying in response to threats to life
I	MEDULA and CORD	Birth	Movement of arms and legs without bodily movement	Birth cry and crying

TABLE 2—*Continued*

		Receptive or Sensory				
Manual Competence		*Visual Competence*		*Auditory Competence*		*Tactile Competence*
Using a hand to write which is consistent with the dominant hemisphere		Reading words using a dominant eye consistent with the dominant hemisphere		Understanding of complete vocabulary and proper sentences with proper ear		Tactile identification of objects using a hand consistent with hemispheric dominance
Bimanual function with one hand in dominant role	Stereopsis	Identification of visual symbols and letters within experience	Stereophonetics	Understanding of 2000 words and simple sentences	Stereognosis	Description of objects by tactile means
Cortical opposition bilaterally and simultaneously		Differentiation of similar but unlike simple visual symbols		Understanding of 10 to 25 words and two word couplets		Tactile differentiation of similar but unlike objects
Cortical opposition in either hand		Convergence of vision resulting in simple depth perception		Understanding of two words of speech		Tactile understanding of the third dimension in objects which appear to be flat
Prehensile Grasp		Appreciation of detail within a configuration		Appreciation of meaningful sound		Appreciation of gnostic sensation
Vital release		Outline perception		Vital response to threatening sounds		Perception of vital sensation
Grasp reflex		Light reflex		Startle reflex		Babinski reflex

From birth to the age of three years in the superior child, six years in the average child, and eight years in the slow child, these skills develop in a predictable schedule. The individual who performs these functions at a level in keeping with his neurological age is considered to have adequate neurological organization. That is to say his neurological age is equal to his chronological age. In the absence of peripheral causes, inability of the individual to perform one or more of the six functions at a level equivalent to his chronological age indicates a lack of neurological organization. In the concepts and experience of the institutes, a neurological age which falls below chronological age is indicative of neurological disorganization and therefore of the presence of brain injury. The functional level of the brain which is involved is shown by the limitations of the individual's performance. The greater the number of functions involved, the more extensive is the injury. The farther neurological age falls below chronological age, the more severe is the injury. The relationship between neurological age and chronological age is presented in Figure 2, and the scale for determining neurological age is given in Table 3.

By scoring a child's performance on each of the 42 criteria and then converting this raw score into its neurological age equivalent, one can determine a child's neurological organization relative to that of his chronological age peers. Using the profile as the diagnostic instrument, one is then able to generate a specific nonsurgical program of remediation.

It is useful to stop at this point to review a little more of the theoretical basis of Doman and Delacato's work. In line with earlier material, they claim:

> As in other animals, the human brain is anatomically symmetrical. This symmetry applies to function as well, except in the highest levels of the human cortex where a distinct asymmetry in function develops. As a result one hemisphere becomes dominant and controls the three motor skills and analyzes the information received by the three sensory facilities which have already been discussed. Full neurological organization includes the development of hemispheric dominance which normally resides in the same hemisphere for all six functions. Pavlov spoke of the "plasticity" of the function of the brain. At the Institutes we regard as fortuitous the anatomic and basic functional symmetry of the

TABLE 3

The Institutes for the Achievement of Human Potential Scale for Determining Neurological Age

Score from Doman-Delacato Profile	*Neurological Age*	
6	0	Months
7	0.5	"
8	1	"
9	1.5	"
10	2.5	"
11	3.5	"
12	4.5	"
13	6	"
14	7.5	"
15	9	"
16	10.5	"
17	12	"
18	13.5	"
19	15	"
20	17	"
21	19	"
22	21	"
23	23	"
24	26	"
25	29	"
26	32	"
27	35	"
28	38	"
29	41	"
30	44	"
31	48	"
32	52	"
33	56	"
34	60	"
35	64	"
36	68	"
37	72	"
38	76	"
39	81	"
40	86	"
41	91	"
42	96	"

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Conversion Table, Form IAHP 011 (Rev. Mar. 15, 1965).

> brain which makes possible transfer of function from a particular area in one cerebral hemisphere to its exact counterpart in the contralateral hemisphere. Essential to the success of such transfer of function are (1) the functional integrity of the hemisphere to which function is being transferred and (2) the presence of what Penfield terms the "uncommitted cortex," functionally capable of accepting the transfer.
>
> The injured brain is never entirely a dead brain. While there may be irrevocably dead cells in the area of injury, there are also cells which are alive but whose function has been depressed as well as uninjured collateral cells which have not yet become functionally involved. The combined effect of cellular death with cellular functional depression creates a picture which is due to lack or to disruption of neurological organization. It fails to reflect the potential which the brain may achieve by means of stimulation directed toward raising the living but functionally depressed cells and the collateral cells to their full capability [Institutes for the Achievement of Human Potential, 1965, p. 5].

At birth man's central nervous system has acquired function up to the neurological level of the spinal cord and medulla from which it ascends progressively through the levels of pons, midbrain, and cortex. The development of sensory pathways precedes those of the corresponding motor tracts. This fact is fundamental to the concepts upon which the institutes base their treatment of brain injury. Great emphasis is placed on the environment as being the source of sensory stimuli, involving touch, pressure, temperature, and visual, auditory, kinesthetic, and proprioceptive sensation to which the individual reacts with a motor (behavioral) response. They have assumed that environmental stimuli of ordinary intensity and duration are inadequate to elicit responses from the functionally uninvolved and depressed cells of the injured brain; however, with great increase of these stimuli in intensity, frequency, and duration, these cells will respond. Therefore, they claim that they do not treat eyes or ears, arms or legs, individual muscles or muscle groups. They claim that they reach the brain itself by pouring into the afferent system of the individual all of the stimuli normally provided by his environment; but they do this with such intensity and frequency that the process will draw, ultimately, a response from the corresponding motor systems. This is the very heart of the treatment procedures.

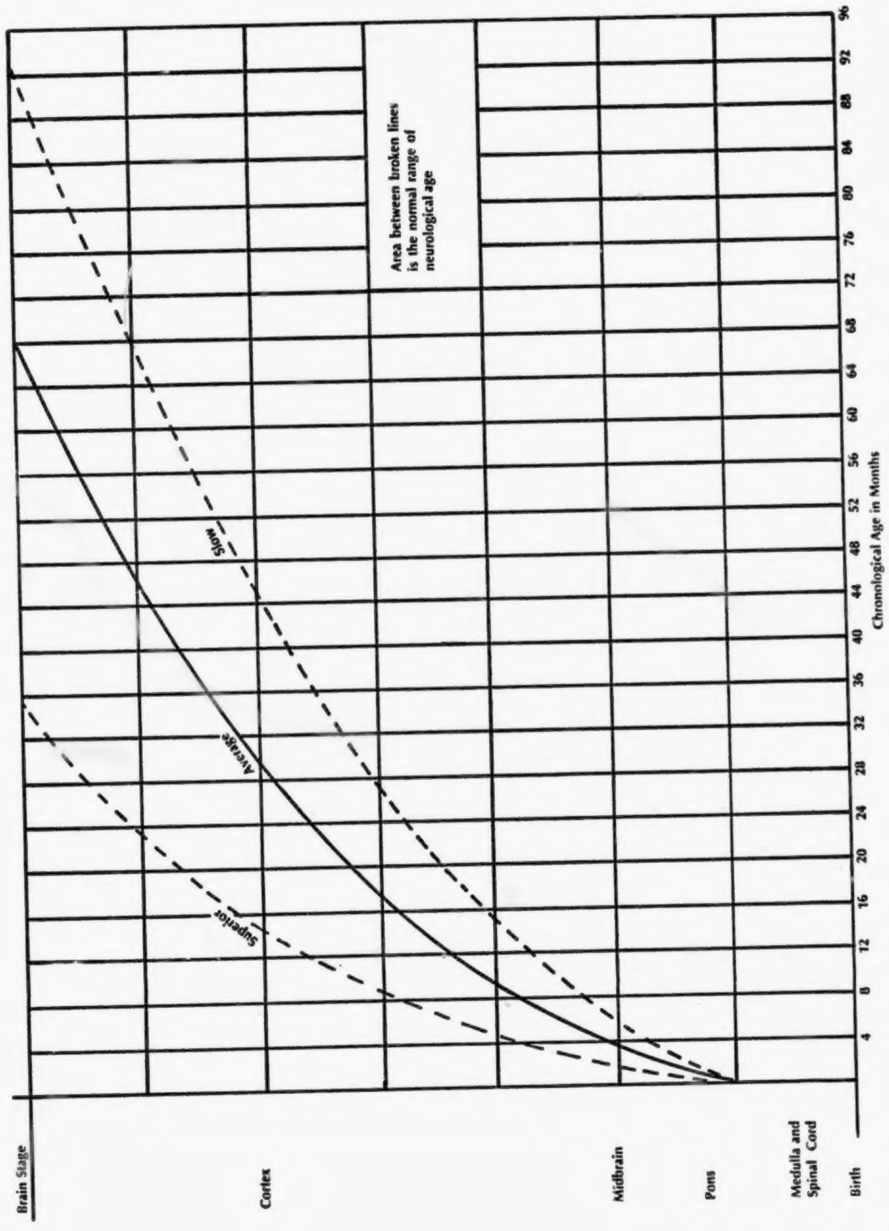

FIGURE 2. *Graphic Representation of Neurological Age*

Treatment Procedure

As mentioned earlier, the work of the institutes involves two broad treatment categories: (a) surgical treatment of the brain, and (b) nonsurgical treatment of the brain utilizing principles of neurological organization.

This section of the discussion on treatment procedures will be restricted to the treatment of the brain by simple nonsurgical methods.

The institutes claim that nonsurgical methods of treatment of brain injury cannot succeed if active or increasing pathology is present in the brain. Of the thousands of brain injured children who are referred to the institutes, only 1.3 percent demonstrate the presence of such pathology at their first evaluation, requiring further neurological or neurosurgical study. The vast majority of brain injured children who are candidates for nonsurgical treatment at the institutes are either postsurgical (32 percent) or are not candidates for surgical intervention at the time of referral (68 per cent) (The Institutes, 1965).

There are five principles (The Institutes, 1965) of nonsurgical treatment of brain injury. These principles are based on the premise that the function of the brain is to relate the organism to its environment. Utilizing each of these principles, the researchers have established groups of effective procedures for the treatment of brain injury. Each procedure encompasses a large number of techniques:

1. Procedures which supply basic discrete bits of information to the brain for storage.
2. Procedures which program the brain.
3. Procedures which demand an immediate response from the brain to a basic discrete bit of information which has just been supplied to the brain.
4. Procedures which permit the brain to respond to previous programing.
5. Procedures which provide an improved physiological environment in which the brain may function.

It must be remembered that all brain pathways can be divided into two broad categories. These are (a) sensory (afferent) pathways which bring information into the brain and (b) motor (efferent) pathways through which the brain reacts by commanding motor responses to the information it has received.

All incoming sensory or afferent pathways are one way roads into the brain and are incapable of carrying an outgoing message. All outgoing motor or efferent pathways are one way roads out from the brain and are incapable of carrying a message into the brain. This is a long recognized and well known fact of neurology which seems to have been completely overlooked in conventional rehabilitation of brain injured patients. Until recent years, classical methods have treated the brain injured patient in purely motor terms. The result of such motor or efferently oriented treatment has been that whatever information the brain has managed to receive has been both accidental and incidental. [*Editor's Note:* It must be noted here that the presence of afferent neurons imbedded in striate muscle provides the anatomical basis for the feedback loop, making it clear that *some* afferent activity is inevitable in any motor performance.]

Between the sensory and motor pathways, deep within the central nervous system, lie the integrative areas, as yet inadequately defined and poorly understood.

The normal cybernetic functioning of the brain is completely dependent upon the integrity of all of these pathways. The total destruction of all motor or all sensory pathways will result in total lack of functional performance of the human being. The partial destruction of one or the other will result in partial lack of functional performance of the individual.

Such lack of functional performance will continue until the former specific pathways are restored to function or until new pathways are established which are capable of completing the total cybernetic loop.

In the human organism, this loop, which begins in the environment, follows sensory pathways to the brain and motor pathways from the brain back to the environment.

All efforts in treatment of the brain injured patient must therefore be directed at locating the break and again closing the circuit.

All of the treatment procedures can be placed within one of the five principles which have already been described.

First Principle: Those procedures which supply basic discrete bits of information to the brain for storage. All of these procedures are entirely sensory in nature and do not anticipate a motor response. They are intended purely to supply the brain with bits of information which are in themselves random. It is not possible to extract either function or information from a brain which has none. Such a brain is in a zero state and will remain so until information is supplied. These procedures provide basic sensory stimuli which range from such simple information as the presence of light, sound, or feeling (as differentiated from the absence of light, sound, or feeling) to much more sophisticated bits of information such as reading a word, hearing a word, or feeling a specific object.

There are only five pathways (all sensory or afferent) through which the brain can gain information, whether in the lowest state of human development or in the highest. These five means are seeing, hearing, feeling, tasting, and smelling. The first three—seeing, hearing, and feeling—are supremely important to complete human function. The last two are most important to humans only in the earliest months of life and become less important with continuing development. These first principle procedures are:

Procedure One: Supplying basic, discrete bits of visual information to the brain in keeping with the individual's present state of visual competence and in anticipation of his next higher level.

Procedure Two: Supplying basic, discrete bits of auditory information to the brain in keeping with the individual's present state of auditory competence and in anticipation of his next higher level.

Procedure Three: Supplying basic, discrete bits of tactile information to the brain in keeping with the individual's present state of tactile competence and in anticipation of his next higher level.

Procedure Four: Supplying basic, discrete bits of gustatory information to the brain in keeping with the individual's present state of gustatory competence and in anticipation of his next higher level.

Procedure Five: Supplying basic, discrete bits of olfactory information to the brain in keeping with the individual's present state of olfactory competence and in anticipation of his next higher level.

The techniques for supplying such basic, discrete bits of information to the brain are geared precisely to the patient's developmental stage in the particular area of sensory competence which is being treated. The patient's level of competence is determined and he is supplied all input normal to that level.

He is then supplied with all sensory input normal to the next higher level which he is unable to accomplish due to brain injury or environmental deprivation. However, in supplying the next higher level, a carefully planned program of greatly intensified and enriched auditory, visual, tactile, gustatory, and olfactory stimuli is made an integral part of the response provoking environment. This is accomplished by increasing such stimuli in frequency, intensity, and duration.

As an example, when a child has been traumatically brain injured and has been in a coma for an extended period of time—over ninety days and perhaps for many years—such a child has been traditionally provided with life sustaining medical and nursing care in a room kept as quiet and free from response provoking environmental impingement as possible. His bed is in a private room with curtains drawn where silence is enforced, and he is as far from noisy areas as possible. He is handled only when necessary.

Exactly the opposite is required if such a child is to have his chance for recovery. All studies in auditory, visual, and tactile deprivation indicate that a well human being placed in such a sterile environment would degenerate neurologically, physically, and intellectually.

In contrast to this, the principles of neurological organization demand that such a child should be provided with the greatest, rather than the least, impingement from his environment.

As a result of the foregoing, a child in such a coma, immediately following the subsidence of cerebral edema, should be placed in a room which is the center of stimulation in an auditory, visual, tactile, gustatory, and olfactory sense. Such a child is functionally blind, deaf, insensate, and without gustatory or olfactory appreciation.

At the Institutes for the Achievement of Human Potential, such a child's bedside table contains a flashlight, two blocks of wood, a tuning fork, pins, brushes, sniff jars containing various strong smelling but unharmful substances, and a variety of other stimulus producing tools.

In addition to regularly and frequently scheduled periods during which the above procedures are utilized, each professional person who passes the child's room is directed to stop long enough to open the child's eyes and shine the flashlight into his eyes, to strike the blocks of wood together against each other sharply near his ear, to pinch his skin, to stick him gently with the pin, to place the tuning fork on various points, to pass the various aromas contained in the bottles under his nose briefly, and to place on his tongue very small amounts of strong tasting foods, insufficient in quantity for him to choke or aspirate.

Under classical methods of handling, many patients are maintained in such a vegetable like state for many years until they eventually succumb, having survived as live human beings only in a technical sense. When such stimulation as has just been specified is introduced, one frequently sees a patient respond by seeing, hearing, feeling, tasting, and smelling in a matter of days or a very few weeks, even though he may have been in a comatose state for months or even years. In the case described, when bits of discrete information were supplied to a brain which had been previously programed by normal development prior to the accident, a state of consciousness is produced which supplants the previous, virtually enforced unconsciousness.

The same procedures apply to the severely brain injured newborn child who has had no opportunity to see, hear, feel, taste, or smell because of brain pathology. They apply also to the older or less severely brain injured child or adult whose neurological development is halted or delayed by pathology at a higher level. The basic, discrete bits of information supplied to such a child are those indicated by the highest level of accomplishment and the next higher anticipated level which is the level of his present inability to perform.

These levels are indicated on the Doman-Delacato developmental profile. The levels of competence may differ greatly in the various specific areas of sensory intake in a given patient due to the focality or diffuseness of the brain injury. Thus an individual child may be receiving bits of auditory information at an extremely primitive level while receiving bits of visual or tactile information at a very advanced level.

This process of supplying bits of information to the brain by the neurological developmentalist is very similar to the storage of precise bits of information in an electronic brain or computer by the engineer and physicist.

Second Principle: Procedures which program the brain. These procedures are entirely sensory in nature and do not seek a motor response. They differ from the procedures of the first principle in that they do not supply basic, discrete bits of information to the brain but, instead, supply large amounts of related and coordinated information. This information is presented in the same related and coordinated manner in which the totally integrated motor response will later be demanded from the brain in the form of human function.

These procedures range from simple to complex, supplying the lower and more primitive levels of the brain with very basic and primitive programs of sensory input, such as tactile programing of simple crawling movements, the auditory programing of simple vital sounds, and the visual programing of outline perception. To the higher levels of the brain they supply very complex and advanced programs of sensory intake such as the tactile programing of complex walking movements, the auditory programing of human speech, and the visual programing of human writing.

These procedures place great reliance upon the tactile, auditory, and visual pathways which are prerequisite to human walking, talking, and writing. Little reliance is placed upon the gustatory and olfactory pathways. They are:

Procedure Six: Supplying tactile programing for various levels of total human movement. This procedure reinforces the program of sensory input at the highest level of mobility competence attained by the child in order to be confident of his totality of function at that level. It also supplies total tactile programing at the next higher level of mobility competence which he has not yet attained.

The techniques employed consist of passively superimposing on the child's body, reproductions, as precise as possible, of the total patterns of movement involved in bodily mobility at his level of competence, as well as at the next higher level of total mobility which he has not yet attained.

The techniques involved range from imposing the lower forms of movement patterns produced by early brain levels, such as simple trunkal movement, through the successively higher patterns of movement produced by more advanced levels of brain, such as crawling and creeping patterns of movement. They culminate in the highest patterns of movement produced only by the exclusively human cortex which include the various levels of uniquely human walking.

In each case of the tactile programing procedure, the total tactile patterns which are prerequisite to the motor accomplishment of the appropriate level of mobility are supplied. Without such tactile input, normal movement of limbs, crawling, creeping, or walking is impossible.

Procedure Seven: Supplying auditory programing for various levels of total human language.

This procedure reinforces the program of sensory input at the highest level of language competence attained by the child in order to be confident of his totality of function at that level. It also supplies total auditory programing at the next higher anticipated level of language competence which he has not yet attained.

The techniques employed consist of passively superimposing on the child's hearing the total patterns of language involved in speech at the level of competence which he has thus far reached, as well as the next higher level of total language of which he is as yet incapable.

The techniques range from imposing the sounds of the lower forms of language patterns produced by early brain levels, such as crying, through the successively higher brain patterns of sound produced by more advanced levels of the brain, such as vital crying patterns and meaningful sound patterns. They culminate in the highest patterns of sound and language produced only by the exclusive human cortex which include the various levels of the uniquely human ability to talk. In each case of the auditory programing procedure, the total auditory patterns which are prerequisite to the motor accomplishment of the appropriate level of speech are supplied to the child. Without such auditory input, normal meaningful sounds or talking are impossible.

Procedure Eight: Supplying visual programing for various levels of total manual competence culminating in human writing. This procedure reinforces the program of sensory input at the highest level of manual competence attained by the child in order to be confident of his totality of function at that level. It also supplies total visual programing at the next higher anticipated level of manual competence which he has not yet attained.

The techniques employed range from imposing the visual images of manual competence patterns produced by lower brain levels, such as vital release, through the successively higher brain patterns of manual competence, such as prehensile grasp patterns and cortical opposition patterns, to the highest patterns of manual competence produced only by the exclusively human cortex. They culminate in the various levels of uniquely human writing.

In each case of the visual patterning procedure the total visual patterns which are prerequisite to the motor accomplishment of the appropriate level of manual competence are supplied to the child. Without such visual input, normal prehensile grasp, cortical opposition, or human writing is impossible. As an example, in order for a human being to write normally as the ultimate motor act of manual competence he must have been capable of receiving the visual input of reading.

Procedure Nine: Developing cortical hemispheric dominance. This procedure accomplishes the attainment of the final ontogenetic development which is unique to man, cortical hemispheric dominance. It is this development which provides human beings with the ability to deal in symbolic language—in speaking and understanding spoken language and in writing and understanding written language.

Dominance is established in one cortical hemisphere, not only for these functions but also for all skills involving the use of the hand or the foot, such as handling food while eating, picking up objects, throwing, kicking, or stepping up. The control of all such functions by a single cortical hemisphere results in unilaterality so that the individual consistently uses the right eye, right ear, right hand, and right foot, or vice versa, depending on which hemisphere is dominant. Hemispheric dominance is genetically determined but it is subject to influences such as physical trauma or cultural factors.

The techniques employed in making unilaterality are superimposed on proper neurological organization of the preceding levels of brain function. Dominant handedness is established by training in such manual skills as writing, picking up, and manipulating objects such as eating utensils and tools, throwing, and stereognosis.

Dominant eyedness is established by occlusion of the subdominant eye by means of color or polaroid filters, by opaque occluders, and, when chiroscopic feedback (hand-eye relationship) is required for the subdominant eye, the stereo-reader.

Dominant earedness is established by occlusion of the subdominant ear. This encourages greater utilization and training of the dominant ear.

Dominant footedness is taught as a skill by means of training in such actions as kicking, stepping up and down, as well as through athletic activities.

Third Principle: Those procedures which demand an immediate response from the brain to a basic, discrete bit of information which has just been supplied to the brain.

All of these procedures are sensory motor in nature and do demand an immediate motor response. While these basic discrete bits of information are in themselves random, they demand a related response. That is to say, they demand a motor reaction which is responsible and appropriate to the specific sensory input.

The brain level to which the stimulus is addressed may be the medulla, pons, midbrain, or cortex. The stimulus may take the form of visual, auditory, tactile, gustatory, or olfactory. The response evoked will be from the brain level addressed and will take the form of mobility, language, or manual response. Depending on the level of brain addressed, the response may be reflexive, perceptive, appreciative, or understanding in nature.

All of these stimuli will begin in the environment and are initiated by the therapist as part of that environment. They pass through the sensory tracts into the brain, which initiates the motor response, which will in turn pass through the motor tracts en route back to the environment upon which they will have their effect.

Procedure Twelve: Supplying basic, discrete bits of information to the brain in keeping with the individual's present state of visual competence and in anticipation of his next higher level. These stimuli demand an immediate response.

Procedure Thirteen: Supplying basic, discrete bits of auditory information to the brain in keeping with the individual's present state of competence and in anticipation of his next higher level. These stimuli demand an immediate response.

Procedure Fourteen: Supplying basic, discrete bits of tactile information to the brain in keeping with the individual's present state of tactile competence and in anticipation of his next higher level. These stimuli demand an immediate response.

Procedure Fifteen: Supplying basic, discrete bits of gustatory information to the brain in keeping with the individual's present state of gustatory competence and in anticipation of his next higher level. These stimuli demand an immediate response.

Procedure Sixteen: Supplying basic, discrete bits of olfactory information to the brain in keeping with the individual's present state of olfactory competence and in anticipation of his next higher level. These stimuli demand an immediate response.

Fourth Principle: Procedures which permit the brain to respond to previous programing. These procedures are sensory motor in nature and provide an optimal opportunity for the brain to utilize in function the programs which were given to the brain in the procedures of the Second Principle.

Since the programs which were supplied to the brain were often repeated and were precisely coordinated large amounts of related information, the responses which will now be elicited from the brain are holistic and precisely coordinated patterns of function. They include patterns of mobility function, which range from crawling through creeping to the highest levels of human walking; patterns of speech function which range from vital sound through meaningful sound to the highest levels of human speech; patterns of manual competence which range from vital release through prehensile grasp to the highest levels of manual competence in human writing; and patterns of human creativity emanating from a single dominant cortical hemisphere, including creative speech composition, creative writing, and creative manual accomplishments.

These patterns exclude the lowest level of brain and spinal cord which are not responsive to programs but only to single bits of discrete information.

These procedures provide an opportune environment in which to retrieve the specific pattern which is desired. As an example, it is easiest for a human being to crawl on a smooth flat surface. It is difficult to crawl on a textured uneven surface. Thus a smooth flat surface is provided for a human being if the goal is to retrieve a pattern of human crawling which has been programed into the brain.

Procedure Seventeen: To provide an opportune environment in which to retrieve motor mobility patterns which have been programed into the brain in sensory tactile forms.

Procedure Eighteen: To provide an opportune environment in which to retrieve motor speech patterns which have been programed into the brain in sensory auditory forms.

Procedure Nineteen: To provide an opportune environment in which to retrieve motor manual patterns which have been programed into the brain in sensory visual form.

Procedure Twenty: To provide an opportune environment in which to retrieve motor movement patterns from a single cortical hemisphere of the brain which have been previously programed into that hemisphere in sensory tactile form.

Procedure Twenty-one: To provide an opportune environment in which to retrieve creative motor speech patterns from a single cortical hemisphere of the brain which have been previously programed into that hemisphere in sensory auditory form.

Procedure Twenty-two: To provide an opportune environment in which to retrieve creative motor writing patterns from a single cortical hemisphere in sensory visual form.

Fifth Principle: Those procedures which provide an improved physiological environment in which the brain may function.

Procedure Twenty-three: This procedure is of a reflex nature and increases the blood flow to the brain, thus supplying additional oxygen and other nutrients to the brain.

Procedure Twenty-four: This procedure prevents overaccumulation of cerebrospinal fluid and cerebrovascular compression.

Effectiveness of Treatment

Included in the patient population at the institutes are children representing all types and degrees of brain injury, with manifestations ranging from minor reading problems to disability verging on the decerebrate. Some of these patients have had no previous therapy; some have undergone neurosurgical procedures ranging from removal of subdural hematoma through installation of ventriculojugular shunt to hemispherectomy. Some have been treated by rehabilitative procedures representing all of the many and varied approaches to brain injury in use during the past 25 years. In all patients there is a discrepancy between neurological age and chronological age and on that basis the institutes claim that they have been unable to distinguish initially those patients who have previously received any type of therapy from those who have had no therapy at all. According to a statistical study which they made of their patients at the initial evaluation, the patients had grown neurologically at an average of about one-third (35 percent) of the normal, regardless of presence or absence of previous treatment.

They claim that with therapy aimed at establishing neurological organization in the highest possible degree, the rate of neurological growth changes from an average of 35 percent of normal to an average of 210 percent of normal.

It is unfortunate that the members of the institutes have published so little in professional journals. One article did appear in 1960 (Doman, Spitz, Zucman, Delacato, and Doman) and a second article, not yet published, was reported at a recent Professional Familiarization Clinic. At this meeting the researchers provided the information presented in Table 4 on 324 unselected patients who had been seen at the institutes during 1962-1963.

TABLE 4

Information on 324 Unselected Patients Seen at the Institute

Subjects	*Percentage*
93 dropped out of study	28.7
3 died during period of study	.9
228 continued on course during study	70.4
Of the 228 Who Continued	
2 discharged as failures	.9
8 discharged as improved	3.5
24 discharged to Institute of Reading Disability	10.5
3 discharged to longitudinal study	1.3
191 still being treated	83.8

Figures 3, 4, 5, and 6 give a graphic picture of the relative progress made by these 324 subjects during the course of treatment.

This, then, is a brief survey of the concepts and procedures of neurological organization developed by Doman and Delacato and their colleagues. Because of the author's own brief personal exposure to the team and the lack of any extensive literature produced by them, he is reluctant to make very many evaluative remarks. However, because of the stimulating nature of the time spent with these men and in the spirit of the warm, and at times heated, discussion which they welcomed and appeared to enjoy, the author takes this opportunity to get some of his main impressions and reactions out into the open.

Evaluation

Favorable Reactions. I am impressed with the comprehensive nature of their theoretical system and the way in which they

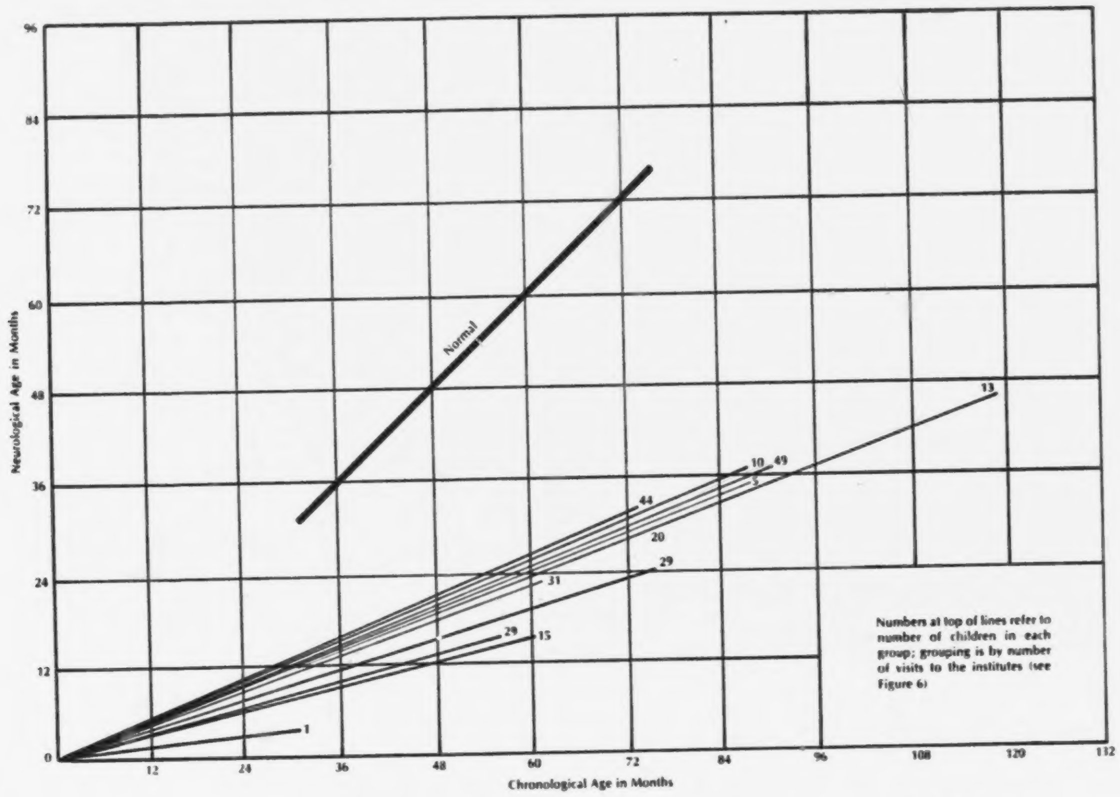

FIGURE 3. *Rehabilitative Progress in 324 Children, in Random Groupings, Ranging from No Treatment to Intensive Treatment by Classical Means Prior to Onset of Treatment by Neurological Organization*

have used it to generate treatment-relevant diagnostic criteria. I lack the ability to evaluate the neurological soundness of the theories themselves; however, I feel that their system is so behavioral in nature that we in psychology and education could utilize their ideas without undertaking a medical degree.

I am particularly impressed with the way in which their diagnosis, treatment, and evaluation is built into the Doman-Delacato Profile. One of the great problems associated with so many diagnostic instruments is that they provide no real guidelines for treatment. At this stage I have not been able to find any empirical verification of the link between

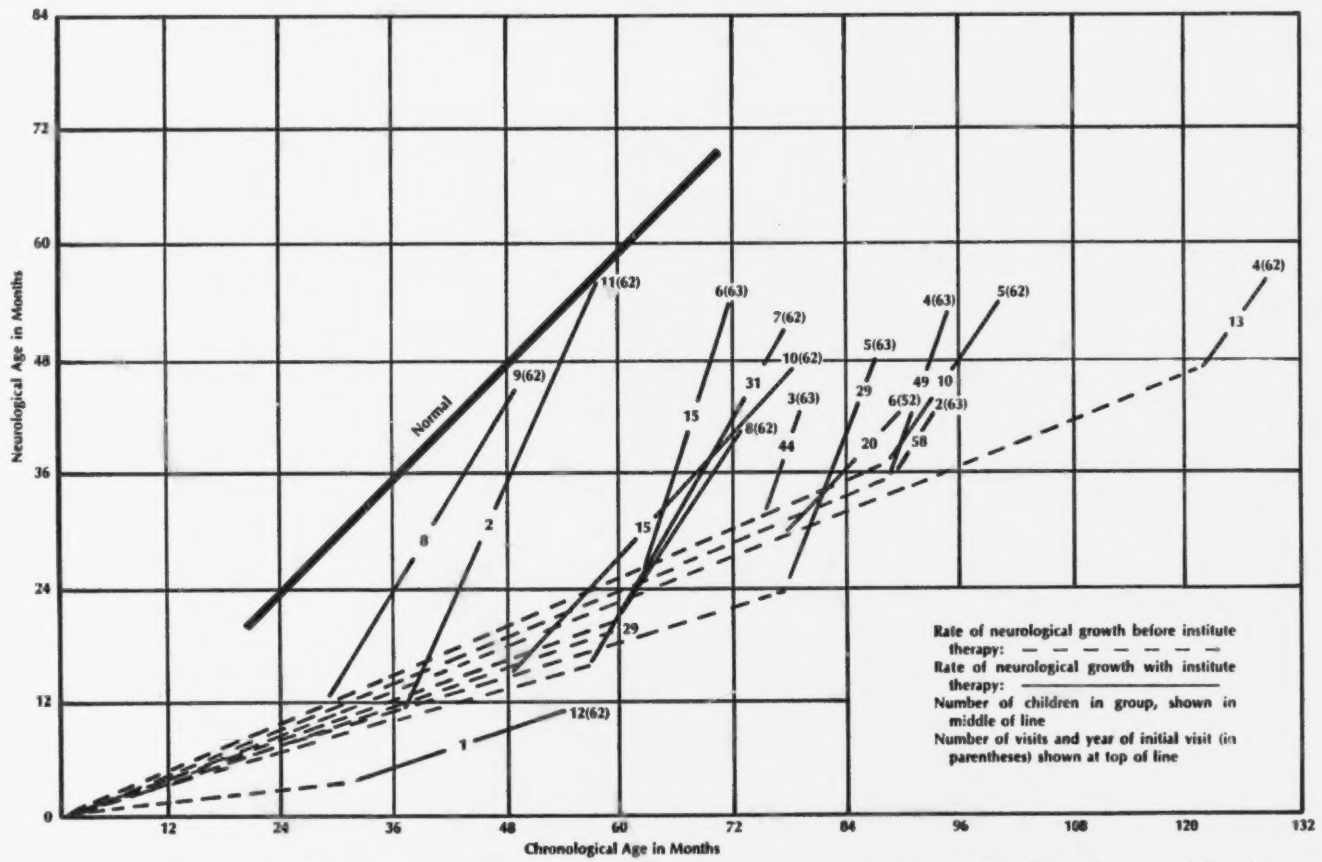

FIGURE 4. *324 Children—Initial Evaluations, 1962 and 1963, Grouped by Number of Visits and Year of Initial Visit*

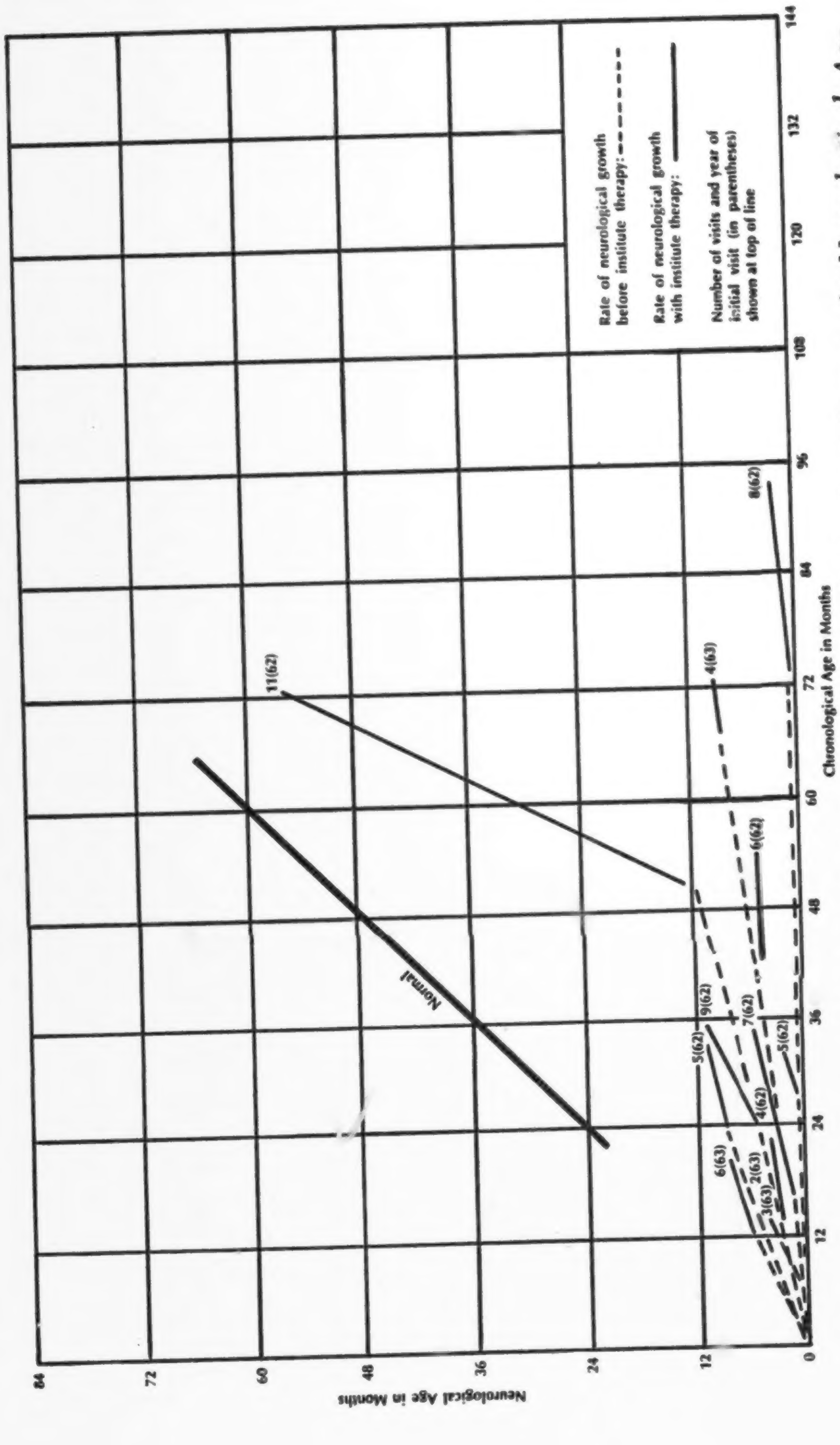

FIGURE 5. *Child (in Each Group) Who Made the Least Increase in Neurological Age*

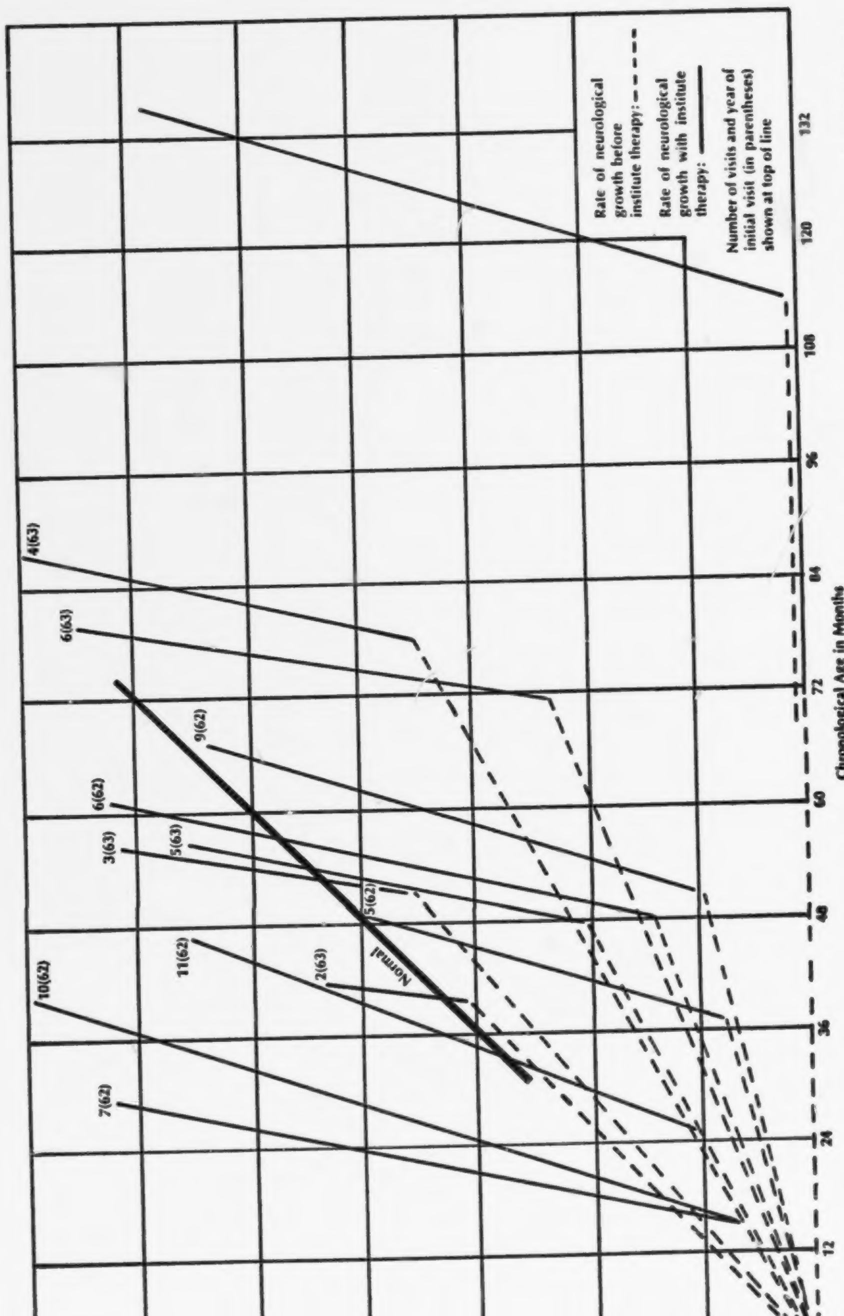

FIGURE 6. *Child (in Each Group) Who Made the Greatest Increase in Neurological Age*

their diagnostic criteria and the specified treatments, but at least these are clearly spelled out and are available for investigation.

One of the strengths of the total program at the institutes seems to be the very successful blending of a number of professional disciplines into a functional unit. I am sure this has led to some role confusion both within and without the system; nevertheless, it has broadened the scope of the service they are able to render and has helped to increase the effectiveness of the overall program.

The way in which they mobilize the local community to work with the child and his family is sound—sound in the sense that it holds the child in the significant social systems of which he needs to be a part, if adequate social, emotional, and intellectual development is to occur. Because of the great demands which are made on parents if they are to provide the necessary experiences for total growth of the impaired child, outside help is essential. The type of sensory and sensory motor experiences required for "normal" development do not need the constant effort and attention of professional persons, but under the guidance of professional consultants, laymen, and even the neighborhood youth can make a significant contribution to the overall development of the brain damaged child.

Finally, I was sufficiently convinced that, for many children, the system works. Children treated in accord with these principles do become functional and do develop the skills of creeping, crawling, walking, running, speaking, reading, and writing. I am sure that far more work needs to be done to delineate the population for whom these procedures are appropriate. I am sure that many have unwisely seen the methods as a panacea for all complaints. Only wide dissemination of both theory and procedures will enable the professional world to determine the extent of the effectiveness of these ideas.

Critical Reactions. One of the effects of blending physical and behavioral scientists into this operation is that the theoretical systems of one discipline are taken over as models by the other disciplines, and the use of these models appears to be hampering the development and articulation of operationally defined concepts in the new areas of research. To be more specific, the work of Doman and Delacato in respect to their developmental profile is strictly at a behavioral, peripheral level and yet they constantly explain what they are doing not in terms of empirically verified, operationally defined concepts, but in terms of the language of their neurological model. Thus, while making peripheral diagnoses and working on peripheral functions, they explain their work in terms of the unobserved and unobservable changes taking place in the brain. In all of their work there is the constant confusion between models and theories—a confusion which could easily blind them to the need for more detailed and systematic verification of their specific ideas and procedures.

Closely tied to this first criticism is their failure to make more systematic use of modern learning theory in refining their training procedures at both the theoretical and empirical levels. So much of what they are doing would seem to be enhanced and made more efficient and effective if the reinforcing variables in the situation were identified and utilized.

While greatly admiring the results which are obtained at the institutes, I was critical of the fact that they see their goals in terms of sensory and sensory motor development and look to the established educational system to provide experiences which will take care of conceptual development. This may be a valid division of labor; however, they failed to convince me that a child treated by their procedures will be able to learn in a normal educational setting with normal instruction.

One of the big criticisms leveled against the group is its failure, up until now, to get sufficient amounts of their data, methodologies, measuring instruments, etc., out into the professional community where their work can be replicated. I am sure that they are taking steps to remedy this situation. As long as they were using medical diagnosis and neurosurgical procedures, extended periods of internship for practitioners were justified, but with the transition to behavioral analysis (Doman-Delacato Profile) and home treatment procedures, prolonged internship for professional persons who wish to replicate their work does seem inappropriate.

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Delacato, C. H. *The treatment and prevention of reading problems.* Springfield, Illinois: Charles C Thomas, 1959.

Delacato, C. H. *The diagnosis and treatment of speech and reading problems.* Springfield, Illinois: Charles C Thomas, 1963.

Doman, G. *How to teach your baby to read.* New York: Random House, 1963.

Doman, R. S., Spitz, E.. B., Zucman, Elizabeth, Delacato, C. A., and Doman, G. Child with severe brain injuries. *Journal of the American Medical Association,* 1960, **174**, 257-262.

Institutes for the Achievement of Human Potential. *Procedural Papers, Professional Familiarization Program.* Philadelphia, Pennsylvania: The Institutes, 1965.

Part 5:
Appendix

Anoxia: An Annotated Bibliography

HARRY LEWIS

Aliab, Eva M. N. The influence of intracranial birth trauma on the subsequent development of the child. *Pediatrica*, 1963, **42**, 7-17. (Russian)

Amos, Elizabeth I. Delayed response performance at three years of age among children with anoxic and non anoxic experiences at birth. *Dissertation Abstracts*, 1960, **20**, 2882.

A delayed response test of 1, 2, 3, 4, 5, and 10 minutes and Stanford-Binet items as an interpolated activity was presented to 19 three year olds with histories of induced breathing and in some cases cyanosis. Twenty controls whose birth records showed normal breathing were used. For the anoxemic youngsters, length of delay related significantly with successful performance. Among controls there was no difference. The two groups differed in respect to mean IQ ($p < .01$). In the anoxic, IQ values related significantly with performance on the three longer delay intervals.

Apgar, Virginia, Girdany, B. R., McIntosh, R., and Taylor, H. C. Neonatal anoxia: a study of the relation of oxygenation at birth to intellectual development. *Pediatrics*, 1955, **15**, 653-662.

No significant correlation was found between levels of blood oxygen content measured at various intervals during the first three hours after birth and intelligence as measured by the Stanford-Binet in early childhood. Low correlation was also found between the Gesell development rating administered at age two and the Stanford-Binet administered at age five.

Ashton, N., and Pedlev, C. Studies on developing retinal vessels. *British Journal of Ophthalmology*, 1962, **46**, 257-276.

It was found that with oxygen (80 percent) vaso-obliteration begins after six hours exposure with capillary closure, together with or soon followed by degenerative changes in endothelial cytoplasm. These results are more in favor of a direct injury to the retinal endothelium than of a perivascular compression.

Bailey, C. J. Interrelationship of asphyxia neonatorum, cerebral palsy, and mental retardation: Present status of the problem. In W. F. Windle (Editor), *Neurological and psychological deficits of asphyxia neonatorum*. Springfield, Illinois: Charles C Thomas, 1958. Pp. 5-30.

There may be a causal relationship between oxygen deficiency at birth and later behavioral abnormalities, such as mental retardation. In order to arrive at this conclusion one must overlook many defects in the studies.

Benaron, H. B. W., Tucker, Beatrice E., Andrews, J. P., Bosher, B., Cohen, J., Froman, Erika, and Yarcorzynski, G. K. Effect of anoxia during labor and immediately after birth on the subsequent development of the child. *American Journal of Obstetrics and Gynecology*, 1960, **80**, 1129-1142.

Children with severe anoxia (not breathing for 30 seconds or more at birth) are more variable than a similar control group. The majority of the children in the anoxic group remain relatively unaffected by severe birth conditions. Many children were of superior ability. However, a greater incidence of feeblemindedness (20 percent as compared with 2.5 percent of the controls) was found, as well as a greater incidence of encephalographic abnormalities (36 percent versus none).

Bradley, C. Organic factors in the psychopathology of childhood. In P. H. Hoch and J. Zubin (Editors), *Psychopathology of childhood.* New York: Grune and Stratton, 1955. Pp. 82-104.

Prognosis for children with organic problems depends on the way in which they are accepted rather than on the degree of organic factors themselves. The psychodynamic reasons for failure and success may well outweigh organic factors. Individuals with cerebral trauma may also develop compensatory skills and defenses which would ameliorate the trauma.

Brander, T. The meaning of below normal birthweight in the development of intracranial birth trauma. *Zentralblatt für Gynakologie,* 1937, **61**, 1645-1648.

As opposed to findings of others, the author finds that more complications exist with lower birth weight. The incidence of mental retardation, word blindness, and epilepsy increases in direct proportion to the drop in birth weight. He ascribes some of the damage to fairly large intracranial hemorrhages caused by a complicated delivery.

Campbell, W. A. B., Cheeseman, E. A., and Kilpatrick, A. W. The effects of neonatal asphyxia on physical and mental development. *Archives of the Diseases of Childhood,* 1950, **25**, 351-359.

All children born during the years 1938 to 1941 with severe asphyxia were followed, along with a group of controls. Seventy-three percent of the above group were reexamined 8 to 11 years later. No significant difference was found in average physical measurements, nor in distribution of intelligence (as measured by Raven's Matrices) between the control group and the asphyxia group.

Commermeyer, J. Neuropathology of asphyxia neonatorum. In W. F. Windle (Editor), *Neurological and psychological defects of asphyxia neonatorum.* Springfield, Illinois: Charles C Thomas, 1958. Pp. 156-172.

There is evidence that anoxia plays an important role in the production of changes in man. We are still unable to say whether the pathological effects of anoxia are direct or produced through an intermediary condition. Anoxia has effects on the visceral organs which may influence the ability of the brain to recover.

Contrevas, M., Donoso, P., and Garnica, H. Traumatism of childbirth and anoxia in the newborn. *Neurocirgraphia,* 1961, **14**, 19-30. (Spanish)

Cooke, R. E. Physiology of asphyxia neonatorum. In W. F. Windle (Editor), *Neurological and psychological defects of asphyxia neonatorum.* Springfield, Illinois: Charles C Thomas, 1958.

Considerable biochemical changes result from anoxia; in addition, marked circulatory changes occur which, if severe, do not permit recovery from the anoxic state. Even after arterial oxygen saturation has been restored rapidly, actual oxygenation of the tissues may be inadequate because of circulatory disturbances. This indicates that the problem of resuscitation of the asphyxiated newborn infant must involve more than simple oxygenation of arterial blood.

Courville, C. B. *Cerebral palsy.* Los Angeles: San Lucas Press, 1954.

A short work (80 pages) outlining the history, etiology, and pathology of cerebral palsy. It describes clinical syndromes and their treatment, and con-

tains many plates showing the pathology of the cortex. The author includes a good bibliography.

Courville, C. B. Antenatal and paranatal circulatory disorders as a cause of cerebral damage in early life. *Journal of Neuropathology and Experimental Neurology*, 1959, **18**, 115-140.

The circulatory (anoxic) genesis of chronic cystic degeneration of white matter is still somewhat in question, as is myelinopathy in early life. An anoxic etiology of structural changes is suggested by the well known vulnerability of the structures to oxygen deficiency. Experimental findings suggest the aberrant location and the hypertrophy of the myelin sheath as well as an increase in their number in the basal ganglia may be the result of a parenchymatous deficit in the regional cortex. The lesion may therefore indirectly result from a primary anoxic or ischemic disorder.

Courville, C. B. Residual changes in the brain incidental to anoxia under general anesthesia. *Anesthesia and Analgesia*, 1960, **39**, 361-368.

Brain tissue changes in a 6 year old child that failed to regain consciousness after anesthesia showed generalized atrophy of the brain on which was superimposed an irregular regional convolutional atrophy. Cortical changes were characterized by laminar necrosis associated with profound parenchymatous damage. It may be presumed that cerebral anoxemia resulted from postanesthetic anoxemia.

Courville, P. Anoxia and its residual encephalitic lesions. *Canadian Anaesthesiology Journal*, 1961, **8**, 3-13.

Courville, C. B. Structural basis for athetosis in cerebral palsied children. *Archives of Pediatrics*, 1961, **78**, 461-474.

Physical damage or specific lesions need not be present to cause a clinical picture of chareo-athetosis; conversely, physical changes in the basal ganglia which during life presented no significant manifestation may be found.

Cross, K. W., Lelong, M., and Smith, C. A. *Anoxia of the newborn infant: a symposium* (Council for Internal Organization of the Medical Sciences). Springfield, Illinois: Charles C Thomas, 1953.

Darke, R. A. Late effects of severe asphyxia neonatorum. *Journal of Pediatrics*, 1944, **24**, 148-158.

There is a continuum in the degree and duration of anoxia. Less severely asphyxiated children do not appear to be significantly retarded while those with severe apnea, even though they show no evidence of injury at birth, are significantly retarded in mental development. There is a combination of degree and duration of anoxia from which the nervous tissues are unable to recover adequately.

Debré, R., Bargeton, E., Mozziconacci, P., and Habib, R. Cerebral lesions due to neonate anoxia. *Archives Françaises de Pediatrie*, 1955, **12**, 673-678. (French)

Dekaban, A., and Windle, F. W. Hemorrhagic lesions in acute birth injuries. *Journal of Neuropathology and Experimental Neurology*, 1962, **21**, 305-307.

A discussion and analysis of the pathogenesis of intraventricular hemorrhage and of intracerebral petechial lesions, it presents preliminary findings which

indicate that petechial and intraventricular hemorrhages in asphyxiated infant monkeys at delivery hardly ever occur.

Denhoff, E., and Robinault, Isabel P. *Cerebral palsy and related disorders: a developmental approach to dysfunction.* New York: McGraw-Hill, 1960.

Cerebral palsy is not a definitive entity in itself, but one aspect of a broader syndrome of cerebral dysfunction. Representing the viewpoint of the Meeting Street School in Providence, Rhode Island, the authors set forth their developmental theories coupled with a team approach. The book contains a list of professional movies and a good bibliography.

Denhoff, E., Smirnoff, V. N., and Holden, R. H. Cerebral palsy. *New England Journal of Medicine,* 1951, **245**, 728-735+.

Early diagnosis of cerebral palsy is possible when the child's development is evaluated at regular intervals and supplemental diagnostic techniques are properly utilized. Cerebral palsy should be regarded as part of a brain damage syndrome rather than as a separate disease entity. A holistic approach is recommended for habilitation, including medical, occupational, and speech therapy as well as special education and vocational guidance. An understanding of the emotional handicaps of cerebral palsy patients and their parents in combination with proper psychiatric guidance is an essential part of therapy. The article has a good bibliography.

Gastelum, B. J. Immediate and late sequels of fetal and neonatal anoxia. *G.A.C. Medecine Mexico,* 1960, **90**, 869-877. (Spanish)

Genn, M. M., and Silverman, W. A. Mental retardation of ex-premature children with retrolental fibroplasia. *Journal of Nervous and Mental Diseases,* 1964, **138**, 79-86.

Fifty-three pairs of retrolental fibroplasia (RLF) cases were matched for birth weight, sex, and race with a group of controls, and a battery of psychological tests was administered to them at age seven and a half. The mean IQ for the RLF children was significantly lower. RLF was not invariably associated with mental retardation. Many RLF children, both sighted and nonsighted, received average and above average IQ scores.

Graham, Frances K. Behavioral differences between normal and traumatized newborns: part I. Test procedures. *Psychological Monographs,* 1956, **70** (Whole No. 427).

Five test procedures–pain threshold test, maturation scale, vision scale, irritability rating, and muscle tension—were developed to provide a means of differentiating normal newborns from those that have been traumatized and possibly brain injured.

Graham, Frances K., Ernhart, Claire B., Thurston, D., and Croft, M. Development three years after perinatal anoxia and other potentially damaging newborn experiences. *Psychological Monographs,* 1962, **76** (Whole No. 522).

Anoxia does not appear to have an all-or-none effect. While it is statistically significant, the prognosis for the individual child is not worsened if he has suffered the degree of anoxia represented by most children. The prognosis does become of concern when the number of or the severity of perinatal complica-

tions increases. This indicates that the concepts of minimal brain damage and of a continuum of reproductive casualty appear reasonable.

Graham, Frances K., Ernhart, Claire B., and Thurston, D. The relationship of neonatal apnea to development at three years. *Research Publication, Association for Research in Nervous and Mental Disease*, 1962, **39**, 159-168.

A prognostic scale taking into account the clinical data of the total perinatal period was used to identify newborns in the poorest condition. That group for whom the prognosis was most guarded was the group which was most impaired. A study of this type may establish an association between perinatal events and three year status, but it cannot establish that the association is a causal one.

Graham, Frances K., Ernhart, Claire B., Croft, M., and Berman, P. W. Brain injury in the preschool child: Some developmental considerations. *Psychological Monographs*, 1963, **77** (Whole No. 573).

Procedures were developed to measure vocabulary skill, conceptual ability, perceptual motor ability, and personality characteristics of preschool children. Particular procedures were selected either because they had successfully differentiated brain impaired from normal adults or because they measured functions relevant to theoretical questions concerning the brain injured child. The effects of age, sex, status group, and interactions were determined.

Graham, Frances K., Eichnor, P. L., Marshall, Joan M., and Thurston, D. Brain injury in the preschool child: Some developmental considerations. *Psychological Monographs*, 1963, **79** (Whole No. 574).

The performances of normal preschool children were compared with those of 70 children diagnosed as brain injured; the presence of injury to the brain was confirmed at the time of testing. All of the 70 had an IQ of 50 or above, and 55 of them were above IQ 69. Brain injured children were significantly but not equally impaired in all areas measured. Personality functioning was sigficantly less affected than nonpersonality. It was suggested that there are systematic differences in the effects of injury depending upon age at the time of injury.

Graham, Frances K., Caldwell, Bettye M., Ernhart, Claire B., Pennoyer, Miriam M., and Hartman, Alexis F. Anoxia as a significant perinatal experience. *Journal of Pediatrics*, 1957, **50**, 556-569.

This is a review of major findings from several studies designed to test the hypothesis that anoxia is a significant perinatal experience. Design factors which may help to account for the discordant conclusions arrived at by various investigators were pointed out. The article includes a good bibliography.

Graham, Frances K., Matarozzo, Ruth G., and Caldwell, Bettye M. Behavioral differences between normal and traumatized newborns: part II. Standardization, reliability, and validity. *Psychological Monographs*, 1956, **70** (Whole No. 428).

Five tests—pain threshold, maturation scale, vision scale, irritability rating, and muscle tension rating were administered to 265 normal infants and 81 infants suffering from anoxia and mechanical birth injury. All tests selected identified some traumatized subjects as abnormal while false positives ranged

from 1 to 3 percent. The percentage identified as abnormal increased with the seriousness of the trauma.

Graham, Frances K., Pennoyer, Miriam M., Caldwell, Bettye M., Greenman, M., and Hartman, Alexis F. Relationship between clinical status and behavior test performance in a newborn group with histories suggesting anoxia. *Journal of Pediatrics*, 1957, **50**, 177-189.

A pain threshhold test, a maturation scale, an irritability rating, and a muscle tension rating were administered to 60 anoxic infants and 62 normal controls. The degree of impairment in test performance reflected the seriousness of the clinical condition. The tests appeared to provide an objective, quantitative, and sensitive method of assessing the clinical status of a newborn infant.

Hannes, W. H. The question of the relationship between asphyxiation and difficult birth in relationship to future physical and neurological problems. *Zeitschrift für Geburtshilfe und Gynakologie*, 1911, **68**, 689-712.

In the light of this investigation at the polyclinic, children born with asphyxia and/or artificial help (forcep extraction) developed as well as those children born naturally without artificial aid. Once an asphyxiated child has lived through the first week there is no further danger.

Hicks, S. P., Cavanaugh, Marie C., and O'Brien, Elaine D. Effects of anoxia on the developing cerebral cortex in the rat. *American Journal of Pathology*, 1962, **40**, 615-635.

Infant rats and mice were immersed in pure nitrogen for 30 minutes. The anoxia had an inhibiting effect on maturation and growth of nuclei in young nerve cells, the suppression of RNA, propagation in dendrite cytoplasm and the stunting of dendrite growth. This is thought to be caused by a lack of oxygen which inhibits the incorporation of amino acids which is coupled to ATP (adenosine triphosphate) production.

Hicks, S. P., Coy, Marie A., and Drago, Elaine R. *Effects of anoxia on the formation of dendrites in the developing cerebral cortex.* U. S. Atomic Energy Commission, Division of Technical Information Extension, 1960, Report No. 6159, 1-8.

Asphyxia can interfere with the cortical development of dendrites whose intricate arrangement seems to have much to do with the discriminating powers of the higher functions of the cortex. The authors also wonder if anoxia may not produce transient or permanent effects on the adult nervous system, before now unsuspected. This process may involve protein synthesis and thus RNA.

Holden, R. H. A review of psychological studies in cerebral palsy, 1947-1952. *American Journal of Mental Deficiency*, 1952, **57**, 92-98.

A review of psychological studies indicates that too much emphasis is placed on intellectual evaluation. The areas of personality evaluation, parent-child relations, and basic psychological research have not as yet been systematically investigated. The author suggests that this is where future research emphasis ought to be. The bibliography has 43 references.

Hughes, S. G. *Symposium on brain damage in children, pediatric clinics of North America.* Philadelphia: W. B. Saunders, 1957.

Hurst, W. E. Experimental demyelination in relation to human and animal diseases. *The American Journal of Medicine*, 1952, **12**, 547-560.

Experimental evidence points to many ways in which demyelination can be produced. Demyelination is merely the response of the white matter to noxious stimuli of a certain intensity. It is quite inconceivable that there is a common basis for all naturally occurring demyelinating diseases of man and animals. The bibliography contains 105 items.

Jacobson, H. N., and Windle, W. F. Responses of foetal and newborn monkeys to asphyxia. *Journal of Physiology*, 1960, **153**, 447-455.

Foetal monkeys with intact membranes were active for a short time after being delivered. The foetal heart rate declined slowly during anoxia. They were successfully resuscitated as long as 6 minutes 54 seconds after the last intra-amniotic gasp. Severe, symmetrical, focal destruction was seen in the brains of monkeys made anoxic as foetuses.

Keith, H. M., and Gage, R. P. Neurologic lesions in relation to asphyxia of the newborn and factors of pregnancy: long term followup. *Pediatrics*, 1960, **26**, 616-622.

Prolonged labor, asphyxia, or delayed respiration at birth do not cause any neurologic abnormalities in children who survive the early months of life. Convulsions are not any more frequent among children who had difficulties at birth than among children who did not have difficulty at birth.

Klebanoff, S. G., Singer, J., and Wilensky, H. Psychological consequences of brain lesions and ablations. *Psychological Bulletin*, 1954, **51**, 1-41.

This is a review of the literature dealing with organic brain damage of the decade preceding 1953. The authors detect a healthy reorientation in emphasis from mere diagnosis and localization to the study of related variables. The literature dealing with psychosurgery indicates results that are disconcerting.

Knobloch, Hilda, and Pasamanick, B. The developmental behavioral approach to the neurologic examination in infancy. *Child Development*, 1962, **33**, 181-198.

The authors present data that the behavioral approach, using the Gesell Developmental and Neurological Examination, is a valid and reliable procedure capable of distinguishing behavior patterns in which abnormalities occur. For detecting minimal brain damage in older children, developmental concepts should be used and new diagnostic tools must be devised.

Kwozek, J. A contribution to the relationship between asphyxiation and difficult birth and later physical and neurological problems. *Zentralblatt für Gynakologie*, 1920, **44**, 983-984.

In the 127 cases that were carefully examined and followed, there were no developmental differences for walking and speaking between those children that had essentially normal births and those that had difficult births (forceps extraction), or were born asphyxiated.

MacKinney, L. G. Asphyxia neonatorum in relation to mental retardation: Current studies in man. In W. F. Windle (Editor), *Neurological and psychological defects of asphyxia neonatorum*. Springfield, Illinois: Charles C Thomas, 1958. Pp. 195-219.

The preliminary data presented suggest the possibility that depressed mental function may be associated with both lower oxygen saturation and clinical distress in newborn infants. Apparently IQ is directly correlated with such variables as clinical signs of distress and O_2 saturation at birth.

Masland, R. L., Sarason, S. B., and Gladwin, T. *Mental subnormality.* New York: Basic Books, 1958.

A thorough, comprehensive overview of the causes of mental retardation, containing chapters on pathological studies, prenatal causes, regeneration of the central nervous system, and surveys of incidence. The book has an excellent bibliography.

Meier, W. G. Delayed effects of natal anoxemia upon behavior and electroencephalographic activity. *Dissertation Abstracts,* 1954, **14**, 1101-1102.

Hypothesis: Natal anoxemia affects a significant portion of the experimental population in (a) poorly adapted responses which may be called stereotyped, (b) retardation in learning complex tasks, and (c) poor acquisition and retention of symbolic behavior. Twenty-six kittens were tested. Of these, fourteen were deprived of oxygen for 30 minutes; the rest were controls. At age six months the kittens were tested with a battery of behavioral problems. With limitations, the results tended to conform to the hypothesis.

Miller, J. A., Jr., Miller, F. S., and Westin, B. Hypothermia in the treatment of asphyxia. *Biologia Neonatorum,* 1964, **6**, 148-163.

Murtagh, J. J., Sagreras, P. O., and Ferrero, R. C. Nervous sequelae of anoxia of the newborn. *Archivos Argentinos de Pediatria,* 1963, **59**, 1-18.

Parmelee, A. H., Cutsforth, G. M., and Jackson, Claire L. Mental development of children with blindness due to retrolental fibroplasia. *American Journal of Diseases of Children,* 1958, **96**, 641-654.

The incidence of mental retardation in retrolental fibroplasia is compared with blindness due to other causes. Any child with an IQ of less than 70 was considered retarded. Thirty-two percent of the retrolental fibroplasia children were considered retarded and 41 percent of the other children were deemed retarded. The difference between the two groups was not considered statistically significant. No findings can be made and more detailed studies of more homogenous groups are advocated.

Pasamanick, B., and Knobloch, Hilda. Brain damage and reproductive casualty. *The American Journal of Orthopsychiatry,* 1960, **30**, 298-305.

Longitudinal studies of development indicate that prolonged prenatal maternal stress at critical periods of fetal development, either by action on the adrenal cortex or through dietary effects, seems to produce death or damage in the offspring. There exists a continuum of reproductive insult, at least partially socioeconomically determined, resulting in a continuum of reproductive casualty extending from death through varying degrees of neuropsychiatric disability.

Pasamanick, G., and Lilienfield, A. M. Association of maternal and fetal factors with development of mental deficiency. *The Journal of the American Medical Association,* 1955, **159**, 155-160.

Records of mentally defective children born in Baltimore between 1935 and 1952 show significantly more complications of pregnancy and delivery, prematurity, and abnormal neonatal conditions than for the matched controls. Bleeding and toxemia during pregnancy seem to be important factors. There is a continuum of reproductive casualty, consisting of brain damage incurred during prenatal and paranatal periods leading to a gradient of injury extending from fetal and neonatal death through cerebral palsy, epilepsy behavior disorder, and mental retardation.

Rosenfeld, G. B., and Bradley, C. Childhood behavior sequelae of asphyxia in infancy. *Pediatrics*, 1948, **2**, 74-85.

Children having pertussis in infancy or asphyxia neonatorum showed the following behavior characteristics from one and a half to two times as frequently as the control group: (a) unpredictable variability of mood, (b) hypermobility, (c) impulsivity, (d) short attention span, (e) fluctuant ability to recall material previously learned, and (f) conspicuous difficulty with arithmetic in school. Five of these behavior characteristics occurred eight times as frequently in the asphyxia group as in the control group.

Schachter, M. Observations on the prognosis of children born following trauma at birth. *American Journal of Mental Deficiency*, 1950, **54**, 456-463.

Asphyxia at birth is more damaging than forceps delivery as far as clinical sequelae are concerned, although the data in this respect are not satisfactory. Of 353 children born following trauma, 48.89 percent had neurological and endocrine manifestations and 35.41 percent had behavior disturbances. It is clear from a comparison with controls that children born of a traumatic delivery show a significantly higher incidence of neurological and behavioral disturbances.

Smith, G. B. Cerebral accidents of childhood and their relationships to mental deficiency. *Welfare Magazine*, 1926, **17**, 18-33.

Skatvedt, M. *Cerebral palsy*. Oslo: Oslo University Press.

Taylor, E. M. *Psychological appraisal of children with cerebral defects*. Cambridge, Massachusetts: Harvard University Press, 1959.

The author gives procedures for the clinical appraisal of severely handicapped children from birth on. By the use of Piagetian developmental concepts, the adaptation of some existing psychological measures, and the design of some new ones, she provides for the evaluation of the mental and physical potential of the child based on clinical and not psychometric criteria.

Thomas, A., and Dargassier, S. A. *Neurological studies of the newborn and the young child*. Paris: Masson et Cie., 1952.

This is a careful, systematic study on the physical development of the new born and very young child, with step by step description of their physical and mental development. The authors give the course and description of disease and how it interrupts this developmental sequence.

Wepman, J. *A selected bibliography on brain impairment, aphasia, and organic psychodiagnosis*. Chicago: Language Research Associates, 1961.

A good bibliography through July, 1964, in the areas of aphasia, agnosia,

apraxia, language development, language impairment, linguistics, and rehabilitation. Organic psychodiagnosis is subdivided into the personality and brain impairment, localization of function, psychometric and projective test studies, psychometric and projective test manuals.

Williams, C. E. Retrolental fibroplasia in association with mental defect. *British Journal of Opthalmology*, 1958, **42**, 549-557.

The incidence of severe mental retardation in retrolental fibroplasia has varied from 10 to 40 percent in different investigations. The incidence of mental retardation in premature children has been variously estimated at between 4 and 10 percent. Even if consideration is given to the fact that most cases of retrolental fibroplasia are extremely premature, the incidence of mental defect is more than is to be expected.

Yeketal, J. Changes in higher nervous activity after hypoxic injury to the brain in the early postnatal period. *Cas Lek Cesk*, 1962, **101**, 656-659. (Czech)

Yeketal, J. Development of the reaction and adaptation of the central nervous system to stagnation, hypoxia, and anoxia during ontogenesis. *Zeitschrift für Arztliche Forbildung*, 1962, **56**, 388-394. (German)

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